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Original Communications

LOWER ESOPHAGEAL WEB

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(by invitation)
Syracuse, N. Y.

In the past when a patient presented himself to the physician, his chief complaint being difficulty in swallowing, diagnostic techniques were primarily focused on confirming or ruling out the presence of carcinoma, impacted foreign body, diverticula, stricture or cardiospasm, etc. Failure to demonstrate pathology of the pharynx, esophagus, or stomach, left the conscientious physician with the logical assumption that the complaints of the patient were on a functional rather than organic basis.

In 1953, Ingelfinger and Kramer[1] published an article in which they discussed 6 patients each of whom was suffering from dysphagia. Endoscopic examinations of these patients were negative but these authors described roentgenologic observations which they referred to as a "contractile esophageal ring." The same year Schatzki and Gary[2] reported similar x-ray findings on 5 of their patients and interpreted them with the term "lower esophageal ring." The true nature of the lesion was at first not clear to these men. But their original work, their subsequent investigation, and our own experience lead us to believe that many of these patients who have had categorical diagnoses of "functional dysphagia" have, in fact, cases of an organic narrowing of the lower esophageal lumen. This so-called "ring" or "web" is responsible for the symptoms of the patient and these same symptoms disappear once the condition is corrected by surgery.

CLINICAL PICTURE

The type and degree of dysphagia conform to and are consistent with the nature of the lesion. The onset may or may not be acute. It is apparently determined by the relative obstructive potential of the food ingested. For

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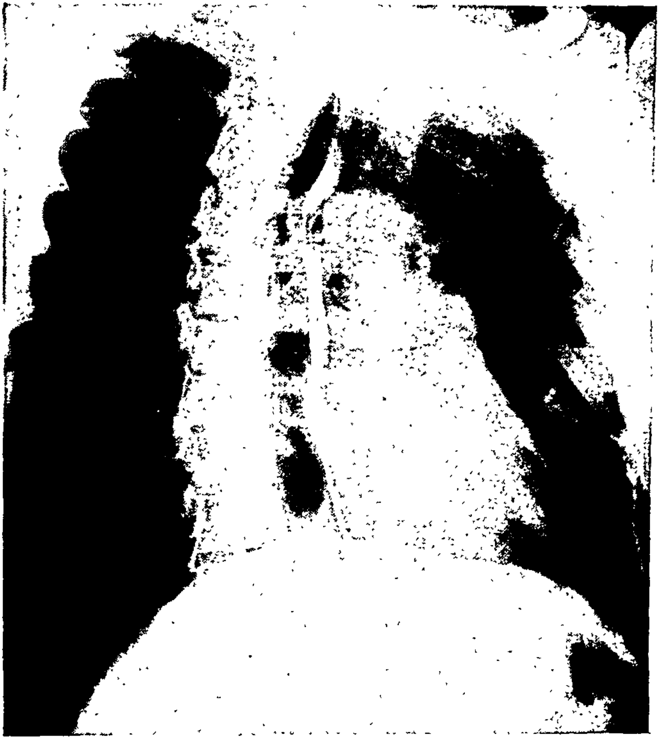

Fig. 1.—Case 1. Thin barium used. Normal-appearing esophagus on first admission.

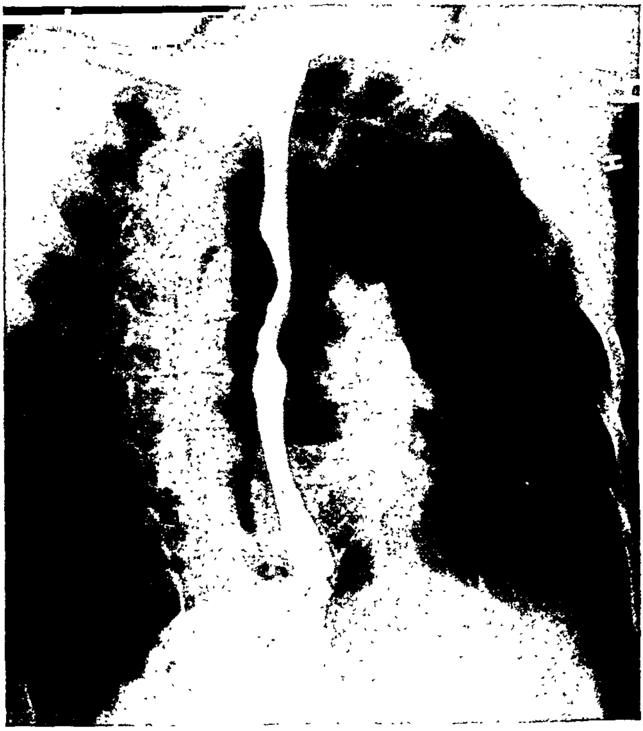

Fig. 2.—Case 1. Thick barium swallow, first admission. Reveals web, but not diagnosed as such.

example, an abrupt change in eating habits from the ingestion of soft food to the ingestion of heavy solids or a poorly masticated bolus may readily produce an acute obstructive episode. Conversely, if the eating habits of the same individual remain unchanged, the onset of dysphagia most likely will be insidious. In either event, once dysphagia has become established, the course is likely to be protracted, of an episodal nature, and may finally become incapacitating.

During the early stages, dysphagia in our cases seemed to have been related to the obstructive potential of the bolus swallowed. Meat and bread were most likely to produce symptoms. Obstruction was associated with low substernal discomfort and a sensation of something being stuck in the lower chest. Drinking water would often alleviate symptoms. For years by careful mastication and scrupulously restricting the diet, severe symptoms were curbed. However, in our cases, dysphagia finally became so severe that intake had consisted essentially of a liquid diet for the year prior to surgery. Neither patient had experienced vomiting, nausea, or heartburn. Both had traveled from one physician to another seeking relief. They were subjected to repeated endoscopic examinations and barium swallows, but such efforts failed to establish a diagnosis. Consequently, the impressive histories and negative findings had led to the frequently mistaken diagnosis of neurosis.

Fig. 3.—Case 1. Thick barium. Second admission six months later. Web not diagnosed but operation advised for obstructing lesion.

ROENTGEN FINDINGS

Figs. 1 and 2 are x-ray pictures showing a barium swallow in the first patient (H. T.) who was experiencing severe dysphagia at the time. It was

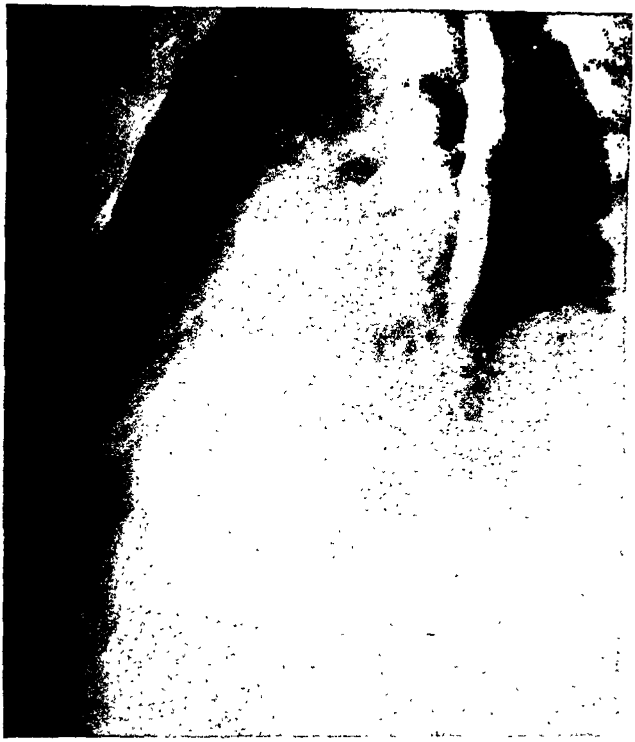

Fig. 4.—Case 2. Thin barium. Interpreted as normal. Marked symptoms at time of examination.

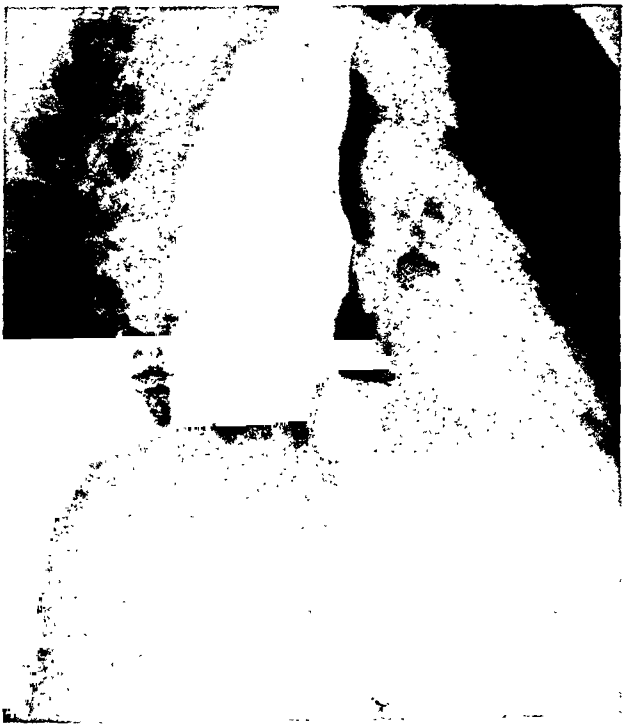

Fig. 5.—Case 2. Thick barium. Second admission, two years later. Web diagnosed.

reported as within normal limits by the consensus of observers, although one man felt sure it represented a stricture. Fig. 3 is a repeat x-ray photograph of the same patient a few months later. A definite weblike structure was demonstrated 5 cm. above the diaphragm and proximal to the esophagogastric junction. Of significance is the fact that the web was not demonstrable until the esophagus above and below it was distended with thick barium. This technique allowed the lumen of the esophagus above and below to exceed the diameter of the lumen of the web.

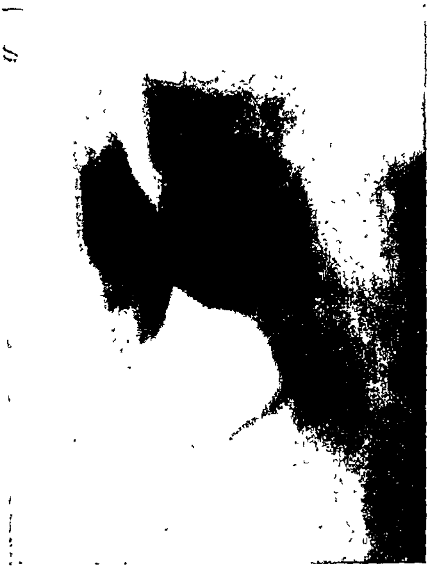

Fig. 6.—Case 2. Spot film of web

Fig. 4 shows an x-ray of a barium swallow in Case 2. Again the original interpretation was that there was no esophageal pathology. Figs. 5 and 6 are x-rays of the esophagus of the same patient two years later. The barium-filled esophagus demonstrates very clearly the weblike structure. Schatzki and Gary clearly describe this lesion in the 5 cases reported in 1953 as a "lower esophageal ring." Their report describes typical x-ray findings although in a subsequent report they were frank to say, "The nature of the abnormality and its origin are not known, but the clinical and roentgenological pictures are characteristic." Our roentgenologic findings demonstrate a thin "web" in the lower esophagus, located 3 to 5 cm. proximal to the esophagogastric junction. It is constant in location and is demonstrable only when the esophagus above and below is distended sufficiently to exceed the diameter of the web. When the esophagus is collapsed the lesion is likely to be missed.

The web is smooth, varying in thickness from 3 to 5 mm. and produces a constant narrowing located centrally, thus producing a "shelflike" symmetrical shadow on the roentgenograms. The lumen in one patient was 3 mm.; it was 5 mm. in the other. The esophagus above the narrowing characteristically is not widened and proximally peristalsis functions normally.

ESOPHAGOSCOPIC FINDINGS

Case 1 had three esophagoscopies performed elsewhere in 1953, each reported as normal. Preoperative esophagoscopy by us in 1954 was reported as essentially normal but the operator experienced difficulty in passing the area interpreted as cardia. The mucosa appeared normal.

Similarly, esophagoscopy of the second patient performed by us in 1952 was recorded as being normal. The instrument was passed into the stomach without difficulty. A second esophagoscopy on this patient was not performed later because repeat roentgenograms were diagnostic of a web.

It is interesting to note that Ingelfinger and Kramer's experience[1] as well as Schatzki and Gary's[2] parallels ours, relative to endoscopic failure. Three of Ingelfinger and Kramer's patients were esophagoscoped and no lesion demonstrated. Four of Schatzki and Gary's were esophagoscoped. The lesion was seen in only one instance.

OPERATIVE AND PATHOLOGIC FINDINGS

At thoracotomy the lesion can easily be demonstrated by opening the esophagus longitudinally a few centimeters proximal to the contemplated site of obstruction and inserting a finger into the esophagus. Once the web has been identified, it is intussuscepted upward by the index finger inserted through a gastrotomy incision (Fig. 8). By internal palpation and this maneuver, the lesion can be easily demonstrated and clearly visualized; with external esophageal palpation the lesion may not be felt. In one case the defect could not be felt externally although a ringlike thickening deep in the wall of the esophagus was palpated.

With a finger in the esophagus, a definite impression was gained that this was a weblike projection into the lumen. The web was leathery in consistency and pliable. The small lumen in both cases (3 to 5 mm., respectively) could barely be felt with the finger tip.

The mucosa above and below the defect felt normal. The obstruction was located above the esophagogastric junction, there being 3 to 5 cm. of normal-appearing esophagus seen below the level of obstruction. In Case 2 a ringlike thickening was present at the base of the web in the esophageal wall.

The elected operative procedure was simple, designed primarily to preserve the integrity of the esophagogastric junction, to alleviate obstruction, and to avoid subsequent stenosis and fibrosis at the operative site. The technique employed is illustrated in Figs. 7, 8, 9, and 10. After the lesion was

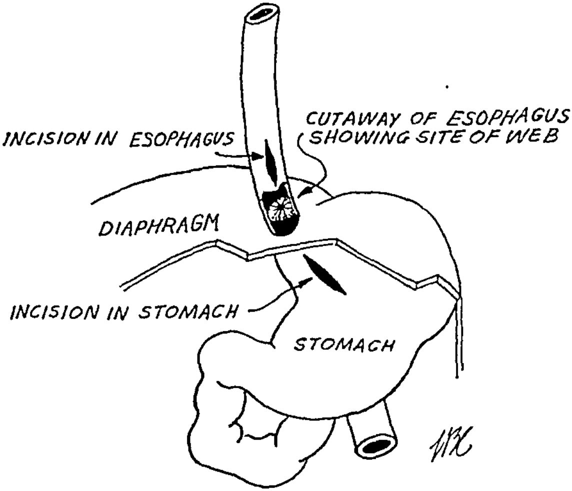

Fig. 7.—Schematic illustration of web in situ with incisions made in esophagus and stomach.

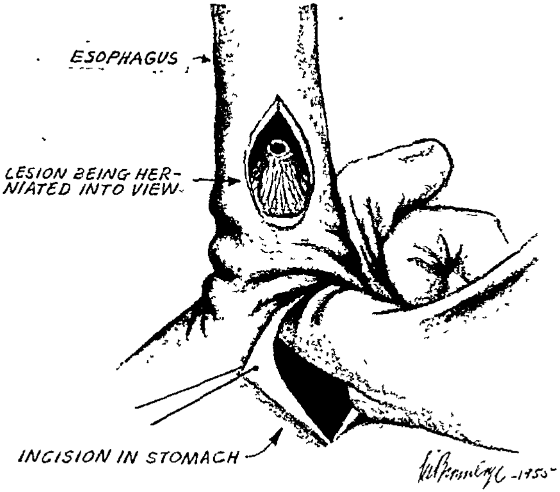

Fig. 8.—Artist's conception of web being intussuscepted upward with left index finger in stomach.

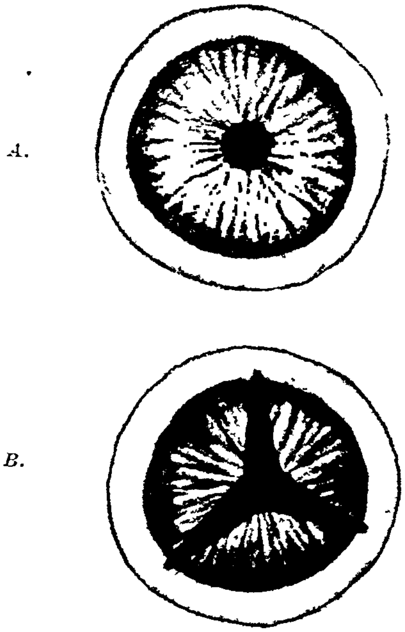

Fig. 9.—Drawing of web and wedges resected. *A*, Cross section of esophagus from above showing weblike membrane. *B*, Cross section of esophagus from above showing wedge-shaped pieces removed from membrane.

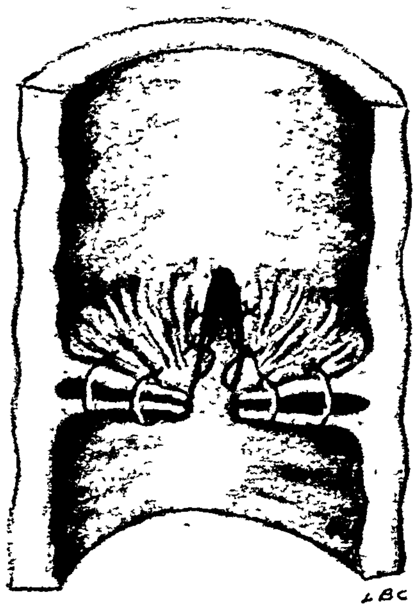

Fig. 10.—Longitudinal section of esophagus showing sutured flaps of weblike membrane. Drawing of repair after wedge resection.

exposed, three pie-shaped wedges of the membrane were removed leaving intervening areas of mucosa at the base to prevent subsequent stricture. The raw areas were sutured with interrupted silk sutures and the stomach and esophagus closed. A Levine tube was then passed into the stomach and the chest closed.

Liquids were started orally on the third postoperative day and solids were given before the seventh postoperative day. To date, the patients are totally relieved of dysphagia, being completely asymptomatic at the seventh and eighth postoperative months, respectively.

PATHOLOGY

At surgery the lesion in each case presented a definite membranous appearance. The esophageal lumina were small, measuring 3 to 5 mm., and were centrally located. The surfaces were smooth having the appearance of normal esophageal mucosa. In each, the web was pliable and felt not unlike the leathery type of a stenosed mitral valve.

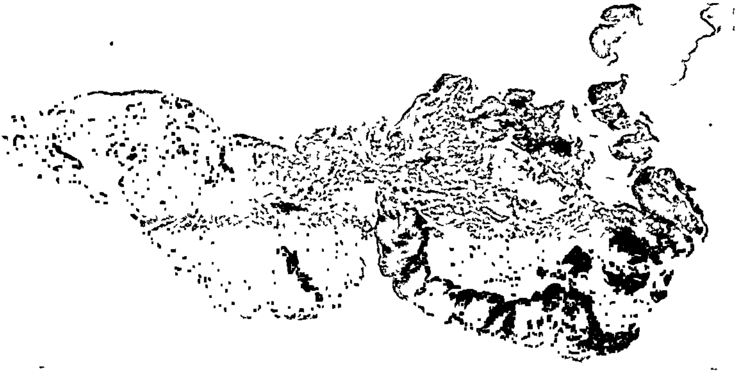

Fig. 11.—Photomicrograph of section of piece of web resected.

Microscopically, the histologic features seen in the wedgelike resected segment were most informative (Figs. 11 and 12). One figure gave a fair indication of the radius of the web although it had decreased in size from fixation. It measured 10 mm. in the fixed state. Squamous-cell epithelium could be seen covering the upper and lower surfaces of the web. There was considerable squamous-cell hyperplasia with folding and piling up of surface epithelium. In many areas associated parakeratosis could be seen. The section clearly demonstrated that mucosa covers the entire surface of the web, although, in one area of this section, a denuded area was seen. The submucosa showed considerable chronic inflammatory cellular infiltration most prominent at the luminal surface where one would expect the most trauma. The outer layer consisted of smooth muscle. At the junction of the web and esophageal wall, smooth muscle could be seen. This abrupt cessation of the mucosa corresponded to the apex of the wedge located in the muscular wall of the esophagus.

The one case reported by Schatzki in which operation was performed revealed similar marked hyperkeratosis and no evidence of ulcer scar or inflammation. Ingelfinger and Kramer's case that was operated upon revealed only thickened muscular wall.

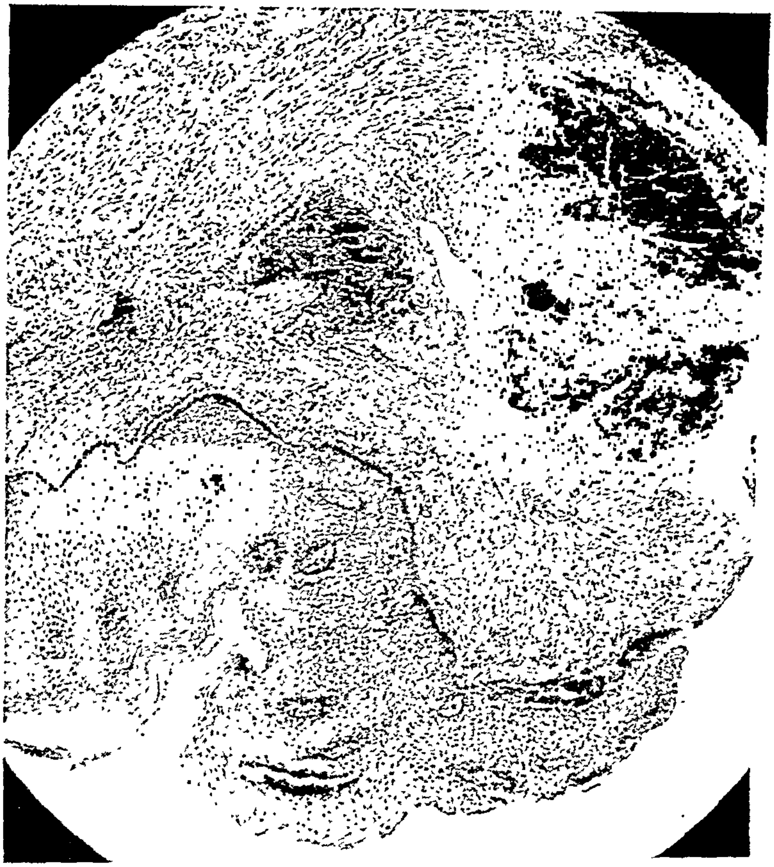

Fig. 12.—Photomicrograph of web biopsy showing hyperkeratosis, inflammation, and circular muscle.

DIFFERENTIAL DIAGNOSIS

The roentgenologic diagnosis of this lesion should not be difficult. It may be deceiving but if the proper diagnostic maneuvers are made, it is less so. The incidence of diagnosis is probably directly proportional to the observer's index of suspicion. With a thin barium swallow, a web may be easily missed. Even when a high degree of obstruction is present, there is very little hesitation of thin barium in passing through the esophagus. A satisfactory examination requires the ingestion of thick barium, in an amount sufficient to fill and thus dilate the esophagus proximal and distal to the web.

The lesion most likely to be confused with a lower esophageal web is an esophageal stricture. Both lesions do not dilate beyond a certain diameter and both may be located in the same area of the esophagus. There are several differential points. A web is symmetrical both circumferentially and longitudinally; the converse is likely to be the case with stricture. The narrowed area relative to a web is likely to be short and thin whereas a strictured area usually is longer and thicker. Further differential points are the lack of delayed emptying and negative endoscopic findings in the instance of the web

Cancer can be quickly eliminated because of the smoothness and thinness of the mucosa and the aforementioned reasons relative to a web. There is no similarity to achalasia. Dilatation proximally, as well as abnormal peristalsis and delayed emptying, is characteristically absent.

CASE REPORTS

CASE 1.—H. T, a 55 year old man, whose first admission to Syracuse University Hospital was on Dec. 22, 1953, with the chief complaint of intermittent dysphagia of four years' duration.

Present Illness—He stated that approximately four years before he began having recurrent mild attacks of having certain foods "stick" in the lower chest Foods most likely to provoke an attack were beef, scalloped potatoes, corn, and bread For two years, symptoms were alleviated by the ingestion of fluids whenever something became "caught." During the third year of his difficulty he controlled symptoms by chewing foods very well and "abstaining from those most likely to cause trouble" Symptoms during the year prior to admission were characterized by more severe obstruction. Many times liquids would fail to relieve symptoms. Occasionally, induced vomiting was necessary. For about a year his diet had been restricted, by his own volition, to ice cream, milk, and cereal. Heart burn, radiating pain, and nausea were absent. There was no weight loss.

After three years of dysphagia, he sought medical advice. Three esophagoscopies and barium studies were done elsewhere. After each examination he was told that he had a "nervous condition" When the situation became desperate, he was transferred to us for evaluation

Physical examination revealed a well developed, well nourished man who was cooperative, intelligent, and did not appear ill. Physical findings were essentially normal.

Esophagoscopy on Dec. 23, 1953, was not remarkable except that at the cardia (50 cm), resistance was encountered. The endoscopist interpreted this as the cardia A biopsy of the mucosa in this area was performed although it appeared to be perfectly normal. It was reported as normal mucosal epithelium.

An investigation of the upper gastrointestinal tract by barium roentgenograms was performed Several roentgenologists agreed that no definite abnormality could be demonstrated. One observer noted a small area of narrowing several centimeters above the cardia. The consensus believed this to be a peristaltic wave, although one observer believed it to be a stricture.

The patient was discharged on Dec. 24, 1953, with a tentative diagnosis of dysphagia of functional origin and with the recommendation that he return for a recheck examination in one month. He returned to his home in northern New York State and was not heard from until July, 1954.

The family physician informed us that the patient's condition had become so severe that the intake of only liquids had recently become ineffectual.

This sequence of events "spelled out" cancer to the patient His wife informed the family doctor that her husband had begun to utter suicidal threats He was then returned to us for further investigation He was readmitted to University Hospital on Aug. 1, 1954. Examination was unchanged from that of the previous admission except for a 5 pound weight loss and an extreme anxiety state.

A repeat barium swallow with a thick mixture revealed a distinct narrowing 5 cm. proximal to the cardia (Fig. 3). Surgery was advised because of the obstructive symptoms although "web" was not diagnosed. Esophagoscopy was not repeated.

On Aug. 2, 1954, at thoracotomy, *no abnormality could be visualized or palpated until* the esophagus had been opened. A 3 cm. longitudinal incision was made proximal to the predetermined site of obstruction. When a finger was inserted into the lumen, a definite obstruction was palpated approximately 3 cm. above the cardia. To obtain accurate visualization and mobilization of this defect, it was decided to open the diaphragm and to make a counter gastrotomy incision. With a finger in the stomach, *the lesion was then herniated* or intussuscepted into the esophageal incision. Excellent control and exposure were obtained (Figs. 7, 8, and 9).

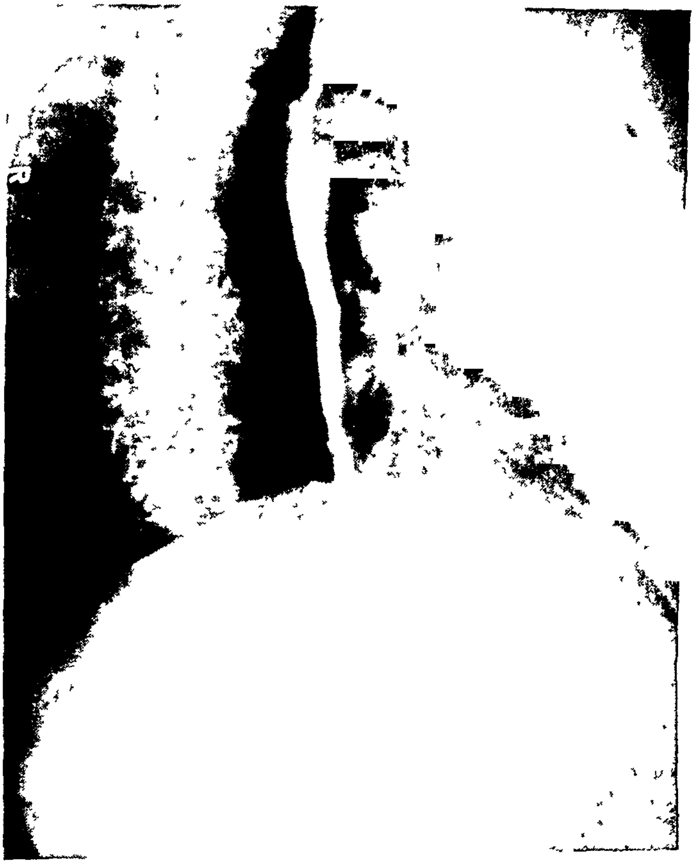

Fig. 13.—Postoperative esophagograms of Case 1.

The obstruction was a definite weblike structure having the characteristics described. A plastic procedure previously described in detail was performed (Figs. 9 and 10).

The postoperative course was uneventful. Postoperative roentgenograms are shown (Fig. 13). The patient has been *completely* relieved of dysphagia and is absolutely free of symptoms. He has returned to a normal diet.

CASE 2.—W. M., a man 55 years of age, entered Syracuse University Hospital on Sept. 12, 1954, with the chief complaint of intermittent dysphagia of seven years' duration.

Present Illness.—The patient stated that 15 years before during an ethanol bout at Thanksgiving, "I almost choked to death after swallowing a large piece of gizzard." He didn't recall further trouble until 1947 when he noted the slow onset of intermittent dysphagia. He recalled having difficulty swallowing large pieces of beef steak and similar large pieces of meat. He noted a sensation of "having a lump in my lower chest," usually relieved by swallowing some liquid. After a year or so, he noted that his eating habits had changed. Chewing food more thoroughly became routine. Strict adherence to this regimen relieved symptoms for several years.

During the two years prior to admission, strict eating habits no longer were effective. For two years his diet was of necessity restricted to liquids, ice cream, and fine cereals.

He was esophagoscoped by us in 1952 but no lesion was found. The stomach was readily entered. Barium studies revealed a normally emptying esophagus (Fig. 4). He was, therefore, told that he had a "nervous condition" and was given pills. He then found that pills would often cause severe symptoms. He denied heartburn, pain, vomiting, and weight loss. He was re-evaluated by us in September, 1954.

Physical examination was essentially negative. The patient was a well-developed, well nourished businessman. He did not appear acutely or chronically ill.

Esophagoscopy was not repeated. Barium study of the esophagus revealed a typical web located just proximal to the cardia. The lumen measured 3 mm. (Figs. 5 and 6).

Fig 14.—Postoperative esophagograms of Case 2.

At exploratory thoracotomy on Sept. 13, 1954, the findings and operative procedure as described in Case 1 were almost identical except that the lesion was palpable before the esophagus was opened. It presented as a firm localized circumferential thickening in the esophageal wall. This area corresponded to the base of the web.

The described plastic repair was performed.

The postoperative roentgenogram is shown in *Fig. 14.*

The patient has been completely relieved of dysphagia and when seen in March, 1955 (6 months postoperatively), he stated, "It's like living anew. I can eat everything [illegible]

COMMENT AND DISCUSSION

Ingelfinger and Kramer deserve credit for first reporting the roentgenographic findings of this lesion, although they were the exact nature of the structure they were describing. They called it a "contractile esophageal ring" and, although 1 case was treated with resection and esophagogastric anastomosis, the pathology of the lesion was not determined.

Schatzki and Gary described accurately the x-ray findings of this lesion and called it a "lower esophageal ring." Although one of their patients was operated on and had an esophagoplasty, they, too, were uncertain about the exact nature of the lesion. We believe that Schatzki and Gary were precise in their original comments and description of this entity as a fixed "ring" which is present at all times although visible only when the esophagus proximal and distal to it is distended more than the maximal diameter of the "ring." Ingelfinger and Kramer believed that the lesion was not preformed, that it occurred as a peristaltic wave approached the lower esophagus, and that the ring might have migrated down the esophagus toward the diaphragm. We are in agreement with Schatzki and Gary's original report and in disagreement with Ingelfinger and Kramer regarding these x-ray findings. However, in a subsequent report, one year later, Schatzki and Gary were inclined to believe that the ring represented the herniated esophagogastric junction. They regarded the narrowing as caused, not by an abnormal muscular contraction, but by an inability of the esophagogastric junction to distend beyond a certain diameter. They stated, "The nature of the abnormality and its origin are not known, but the clinical and roentgenologic pictures are characteristic."[3]

Surgery in our 2 cases has proved the actual identity of the lesion to be a firm, moderately rigid web (or diaphragm) comprised of hyperplastic squamous-cell epithelium, submucosa, and muscularis quite similar to the normal lower esophageal wall. The web is about 3 mm. thick. In the first case the lumen was 3 mm. in diameter, in the second it was 5 mm. It is intraesophageal, about 3 cm. above the esophagogastric junction. In the second case, in addition to the web, there was a thickened intramural ring within the muscular wall the nature of which is not known. Both cases were undiagnosed with the first barium x-ray studies. Both were undiagnosed at esophagoscopy. This may seem faulty and difficult to understand. In each instance the instrument was passed to the obstruction, but it was not visualized as such. The endoscopist thought it was merely a matter of a difficult cardia to pass, since there was no tumor, ulcer, esophagitis, or visible stricture.* Both cases were operated upon in light of the subsequent x-ray findings, plus the severe obstructive symptoms. In the first case, the esophagus appeared normal and was negative to palpation. An esophagotomy was done about 4 cm. above the cardia and the obstruction palpated. The end of a Kelly clamp could be passed through the lumen. With the left index finger introduced through a gastrotomy incision, the web was forced upward into the esophagotomy and a local plastic operation was done. In this manner, the cardia was preserved. In the second case, the web was treated in the identical manner. The coexisting "ring" was incised and fractured. In this case, the ring was palpated externally. Clinically, this simple plastic procedure has proved to be completely effective.

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*With our present knowledge and experience, we think endoscopy will be properly interpreted in future cases of web.

Why does symptomatology occur late in life when the lesion is apparently congenital in origin? This poses an interesting problem and may stimulate considerable speculation. Is there sufficient hyperplasia of the mucosal epithelium and resultant "folding" of the layer to account for a change in lumen size? Does trauma, with associated edema and chronic inflammatory reaction, influence the mobility of the web? Perhaps these changes explain the mechanism of late symptoms. Currently, there are insufficient data to make positive statements regarding these questions. It is hoped that subsequent investigation will supply the answer.

SUMMARY AND CONCLUSIONS

1. Two case reports have been reviewed illustrating a generally unrecognized clinical entity which causes dysphagia.

2. The etiological factor has been clearly established as a membranous weblike structure occurring in the lower third of the esophagus.

3. Its pathologic and clinical characteristics have been discussed.

4. A successful operative approach has been described.

5. Lower esophageal web should be included in the differential diagnosis of dysphagia.

We wish to acknowledge the advisory and technical assistance of Dr. Paul Bunn, Associate Professor of Medicine, Dr. Richard B. Jamieson, Assistant Professor of Bacteriology, and Miss Stella Zimmer, Photography Department, State of New York, Upstate Medical Center, Syracuse, New York.

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DISCUSSION

DR. RICHARD H. SWEET, Boston, Mass.—Dr. Bugden has brought to our attention a most interesting lesion, but I submit that he is confusing two different conditions, because I do not believe that the so-called contraction ring described by Ingelfinger and also by Schatzki is necessarily the same thing. We at Massachusetts General Hospital have been puzzled to explain their findings. I have encountered three actual lower esophageal webs, two in infants and one in an adult, all three operated upon by local procedures such as described by Dr. Bugden and, incidentally, I favor opening the esophagus and doing a circumferential excision of the web.

In the cases that Dr. Schatzki shows us and in Dr. Ingelfinger's cases, one cannot see anything in the esophagus by esophagoscope. The instrument can be passed right into the stomach. On the other hand, these webs, including the three webs I have had, are readily seen by the esophagoscope. In the infants, attempts to dilate them were unsuccessful. There was an obvious mechanical difficulty. Some of the cases that Schatzki calls contrac tion rings, I believe are small hiatus hernias where the esophagogastric junction is a little above the diaphragm, and not infrequently these patients with almost unrecognizable small hiatus hernias may have obstruction when they change their feeding habits, as Dr. Bugden has pointed out. But I think he is talking about two entirely different conditions here today.

DR. RALPH ADAMS, Boston, Mass.—Almost everybody in this Association has a case of almost anything, and I do not want to duplicate the lung abscess experience of a few years ago when practically every youngster present seemed to have one to report. But in the two cases of web of the type pictured, I think that the treatment may be much simpler than that diagramed and exemplified. I am sure there are two different conditions; Dr. Sweet brought that out. They may be distinguished by careful fluoroscopic and x-ray examination. No esophagus has been properly examined until the patient has been fluoroscoped in the Trendelenburg position. If bread is mixed with barium, thin barium becomes the functional equivalent of thick barium. In the Trendelenburg position, if one gives thin barium and then a swallow of thick barium subsequently, lower esophageal lesions, and particularly this one, are made evident more easily and more beautifully than by any other method. Dilatation by means of inflatable bag bouginage will correct these affairs very promptly and in a minimum length of time.

DR. MAX G. CARTER, New Haven, Conn.—I am tremendously interested in this problem and delighted to see that it is again brought to light by Dr. Bugden, but I rather agree with Dr. Sweet that perhaps he may have a different concept of what Dr. Schatzki has brought out from what I have. Since our roentgenologist became aware of this disorder some two years ago we have now seen 4 cases. These cases can be identified from the history, at least to some extent, and when they are examined properly with dilatation of the esophagus during fluoroscopy, the diagnosis becomes clear. In not one of these cases was there any abnormality by esophagoscopy except that the esophagoscope entered the stomach somewhat more readily than is usual. We therefore had the feeling that the patients probably had a "short esophagus" type hiatus hernia. This has been confirmed by x-ray. Finally, these patients have been subjected to dilatation by the Hurst mercury bougies which pass easily through a No. 48 F. without the slightest feeling of resistance. I may be in error, but I cannot imagine that would happen so easily with the cases Dr. Bugden showed us.

(Slide) I am sure many of you have seen this illustration from the textbook by Dr. Templeton. He pointed out that this was a lesion which could be identified when the esophagus was distended, was not malignant, and should not be mistaken for malignancy. That was in 1944. Dr. Templeton did not name the lesion.

(Slide) This illustrates a classical "contraction ring." On the right there is distention of the esophagus with air, almost like a contrast enema, showing the narrowed portion.

(Slide) These pictures are of another patient with similar findings. There is the ordinary esophagram on the left and on the right the esophagus when the patient has swallowed bread together with thin barium. Our roentgenologist has become astute in finding these and I would like to ask Dr. Bugden whether he feels that this sort of lesion should be operated on. We have not operated on any of them. The patients report symptoms occurring during periods of emotional stress, when they perhaps eat a little too fast. One patient would get upset when his teen-age son would take the car and keep it out too late; then for a couple of days he would not be able to eat steak. When he learned what his problem was and that when he was angry with his son he had to chew a little more thoroughly, he could eat normally again.

DR. I. DARIN PUPPEL, Columbus, Ohio.—In our experience, the use of a No. 00 barium-filled capsule has been more positive than the use of barium bread in bringing out defects in the lower esophagus.

DR. B. H. RAMSAY, Los Angeles, Calif.—I should like to report the case of a 61-year-old man who was about to commit suicide. He stated that he had had no difficulty whatsoever with swallowing until eight years before, when suddenly, with a normal meal, he choked and thereafter he had never had anything but barley water and unsweetened gelatine. The correct diagnosis of web obstruction was not made. I operated upon him and did an esophagotomy of the lower 3 inches of esophagus with a Levine tube in place. The esophagus was hypertrophied; the mucosa appeared normal. I asked the anesthetist to pass a Levine tube to the stomach, and it passed the area of narrowing into the proximal inch of the stomach, but no further. In order to investigate this, I inserted my finger into the stomach through a gastrotomy opening and felt a membrane between the finger and the tube. I want to point this out, because this man had had dilations on several occasions by very competent men and each time was told that the esophagus was "like a sponge." Actually, the Levine tube had invaginated a membrane downward into the stomach. There was a dense membrane about 0.5 mm. in thickness with a very small opening in the center. Because these membranes can be displaced or stretched downward, esophageal dilatation may not be simple. If the dilator does not penetrate the opening in the esophageal web but displaces it downward, the dilatation force is directed against the esophageal wall but cannot affect the web itself.

DR. C. F. STOREY, Portsmouth, Va.—I have had one patient with a lower esophageal web identical to those described by Dr. Bugden except that it was located somewhat higher in the esophagus near the junction of the lower and middle thirds. This lesion was encountered in a 9-month-old white girl baby who had had increasing dysphagia and regurgitation since birth. She did fairly well on liquid nourishment but was unable to take any type of solid food. At operation through the right chest the lesion could not be felt through the wall of the mobilized esophagus although it had been accurately localized by preoperative barium swallows. A No. 16 red rubber urethral catheter was passed down the esophagus until the obstruction was met. A 1.5 cm. segment of the esophagus was then resected including of course the esophageal web, and esophageal continuity was restored by a two-layer end-to-end anastomosis. Examination of the resected specimen clearly showed that an operation of unnecessary magnitude had been performed. A small longitudinal incision in the muscle coats of the esophagus, resection of the web, and closure as described by Dr. Bugden would have been perfectly adequate. The patient's postoperative course was entirely uncomplicated and there has been no tendency to narrowing at the site of the anastomosis in the eight months that have elapsed since her operation. The child is now able to eat all foods without difficulty.

DR. RALPH BERG, JR., Spokane, Wash.—I would like to make the following remarks in discussing the paper by Dr. Walter F. Bugden and Dr. J. Ernest Delmonico. Since this lesion is uncommon I wish to present 2 cases diagnosed in childhood.

Slide 1 demonstrates a diaphragmatic obstruction in the distal fourth of the esophagus partially obstructing it. This boy was 23 months old. This second slide demonstrates the findings two weeks following surgical excision of the diaphragm. This is the site of the esophagotomy which you note is the widest portion of the esophagus. Slide 3 shows a radiolucent foreign body obstructing the esophagus by occluding the tiny lumen in a fibrous diaphragm also in the distal fourth of the esophagus. This child was an 11-month-old girl.

Both patients have obtained complete relief after surgical excision of the diaphragms. Both patients had apparently normal esophageal function until they began to take solid food, then intermittent dysphagia appeared with solids. The boy's lesion was missed in two preceding esophagrams. The family had consulted a host of doctors trying to find the cause of his intermittent dysphagia and they were misled by "negative" radiographic studies.

The first child is about two years postoperative, and the second one year postoperative. Neither patient has had dysphagia or obstruction since operation and their diets are entirely normal. In both of these patients, the lesion was readily seen at esophagoscopy and attempts at dilatation were failures. Neither of these children had a history of ingestion of corrosive agents.

Although these radiographs seem to be almost identical to those shown by Drs. Bugden and Delmonico, it seems inconceivable that a lesion such as demonstrated here could remain asymptomatic until adult life. Lesions of this type certainly merit further attention.

DR. ERNEST DELMONICO (Closing).—I wish to thank the discussants.

One purpose in presenting this subject was to stimulate interest, discussion, and investigation. From the comments of the previous discussants, I believe the purpose has been fulfilled. With regard to some of the questions raised, we have reserved some ammunition.

(Slide) A third-year medical student came to us one Sunday afternoon with a foreign body lodged in the lower esophagus. Prior to esophagoscopy he related that for several years he was in the habit of carefully masticating his food to avoid dysphagia. Esophagoscopy located a small bolus of meat lodged near the cardia. Following its removal a definite narrowing was visualized near the esophagogastric junction. An attempt to pass the esophagoscope through this area resulted in fracturing the "web" as represented on this postesophagoscopic barium swallow. The patient no longer has dysphagia. This suggests that in some instances the lesion may be treated by endoscopic means.

(Slide) The next patient was a man 55 years of age who complained of having dysphagia for 16 years. The web in this instance was responsible for only mild dysphagia and therefore a corrective procedure was not performed. This slide again illustrates failure of a thin barium swallow to demonstrate the lesion.

(Slide) This is the same case. However, at this time a barium swallow with a bolus of bread was employed. Notice the relatively large diameter of the lumen of this web. Considerable mobility of the web was observed fluoroscopically, which may be a factor in producing only mild obstruction.

Regarding treatment, we are not certain which cases should be treated by the endoscopic route and which by esophagoplasty. There are many variations of webs with regard to size of lumen thickness, pliability, etc. We do feel that the 2 cases reported in our paper could not possibly have been successfully treated endoscopically.

LYMPHOMA OF THE LUNG AND PLEURA

WILLARD VAN HAZEL, M.D.,* AND ROBERT J. JENSIK, M.D.** (BY INVITATION)
CHICAGO, ILL.

THE related lymphogenous diseases, lymphosarcoma and Hodgkin's disease, when occurring in the thorax have usually been considered to be mediastinal lesions. Pulmonary and/or pleural involvement are seen in these disorders in incidence varying up to 33 per cent (parenchymal) and 50 per cent (pleural) (Rubin[1]) but these localizations usually accompany the widespread dissemination seen in advanced, preterminal, or autopsy material.

In Robbin's[2] analysis of a large series of proved lymphoma cases, approximately 7 per cent were considered to have developed a lymphomatous process in the lungs. This analysis eliminated all pleural fluid cases and conceivably excluded some other cases, which possibly accounts for this low figure.

Isolated organ lymphoma has been reported in bowel and stomach and examples of localized lesions, which would have permitted surgical excisions, were seen in 10 per cent of the autopsy material comprising Gall and Mallory's[3] series of proved lymphomas.

More recently, case reports of pulmonary lymphoma treated by lobectomy or pneumonectomy have appeared. the first of these by Churchill,[4] Spatt, and Grayzel,[5] and Maier (1948),[6] although the resection in the latter report actually was done in 1944.

The patient reported upon by Dr. Churchill was again included in Robbins' series, five and one-half years following resection, at which time a recurrence had developed in the lower lobe and in the left lung.

Subsequent reports have also been published by Anlyan, Lovingood, and Klassen (1950)[7]; Beck and Reganis (1951)[8]; Weissman and Christie (1951)[9]; Sarokhan and Morrison (1953)[10]; and Grimes, Weirich, and Stephens (1954).[11]

Our series consists of 12 cases seen from 1945 to the present time at St. Luke's Hospital and Research and Educational Hospital of the University of Illinois and is divided into seven parenchymal and five pleural types. Brief abstracts of these cases follow although a summary of the data appears in Tables I and II.

CASE REPORTS

CASE 1.—January, 1946. The patient (M. W.) was a 27-year-old white woman who had complained of shortness of breath, a cough, and expectoration of whitish sputum for six months. The onset was acute with pain in the left chest and the symptoms had increased in severity until the time of admission with the additional complaints of sweating, weight loss (25 pounds), and weakness.

From St Luke's Hospital, University of Illinois College of Medicine, Chicago, Ill.

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N J, April 24 to 26, 1955.

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**Associate Attending Surgeon, St. Luke's Hospital; Clinical Assistant Pr gery, University of Illinois College of Medicine

TABLE I. PATIENTS WITH LYMPHOMA INVOLVING THE PULMONARY PARENCHYMA

Lung Parenchyma

PATIENT	AGE	SEX	SITE	X-RAY	HISTOLOGIC DIAGNOSIS	TREATMENT	RESULTS
1. M. W. 9-45	27	F	L. Lung	Uniform density lower ⅔ left lung	Hodgkin's sarcoma	Partial resection of tumor; nitrogen mustard; x-ray	Dead, 3-54; 8½ yr. survival
2. A. F. 3-47	53	F	R. Lung	Circumscribed density, RLL, cardiophrenic angle	Lymphocytic lymphoma	Right pneumonectomy	Dead, 10-50; 3½ yr. survival. Cardiac failure
3. C. S. 1-49	70	M	L. Lung	Circumscribed density, LUL	Lymphocytic lymphoma	Left upper lobectomy	Lost to follow-up 2 yr.
4. W. J. 11-49	53	M	R. Lung	Circumscribed density, RUL	Lymphocytic lymphoma	Right upper lobectomy	Alive—well 5½ yr.
5. I. W. 4-49	24	F	R. Lung	Density—right lung cavitation and fluid level	Reticulum-cell sarcoma	Right pneumonectomy	Alive—well 5½ yr.
6. M. S. -49	25	F	Bilateral	Multiple nodular densities	Hodgkin's disease	Node biopsy; x-ray; nitrogen mustard	Alive 5½ yr.
7. G. C. 11-53	46	F	L. Lung	Circumscribed density, LLL	Lymphocytic lymphoma	Left lower lobectomy	Alive—well 1 yr. 4 mo.

TABLE II. PATIENTS WITH PLEURAL FLUID AS THE PRIMARY ROENTGEN FINDING

Pleural Fluid

PATIENT	AGE	SEX	SITE	X-RAY	HISTOLOGIC DIAGNOSIS	TREATMENT	RESULTS
8. C. D. 7-42	48	M	Right pleura, Left lung	Fluid and density, right chest Density; left lung	Lymphocytic lymphoma	Partial removal; x-ray	Alive—well 13 yr.
9. E. H. 12-47	43	F	Pleural fluid, left	Density, left chest	Hodgkin's disease	X-ray; nitrogen mustard	Dead, 7-51; 3½ yr. survival
10. I. E. 12-49	40	F	Pleural fluid, left	Density, left chest	Lymphocytic lymphoma	X-ray; nitrogen mustard: drainage of empyema	Dead, 2-55; 5½ yr. survival
11. T. M. 10-53	67	F	Right pleura, mediastinum	Pleural density	Hodgkin's disease	Thoracotomy; x-ray; nitrogen mustard	Dead, 2-55; 1⅓ yr.
12. V. D. 11-53	50	F	Right pleura mediastinum	Pleural density Mediastinal enlargement	Lympocytic infiltration of pleura	Repeated aspirations; nitrogen mustard; x-ray	Dead, 4-54; 5 mo.

Physical examination revealed an emaciated, pale, white woman who appeared chronically ill and had a temperature of 102° F There was an enlarged node in the left cervical region and on the left side of the chest dullness was elicited in the lower half Breath sounds were absent in the corresponding area

The laboratory data revealed the red blood count 2 2 million, with a white blood count of 13,900 and hemoglobin, 6 4 Gm Sputum and bronchoscopic secretions were negative for tubercle bacilli

X rays of the chest over a 7 month period revealed a gradually increasing density in the left lung field which, at the time of admission, occupied two thirds of the left chest (Fig 1)

Bronchoscopy was noncontributory and a needle aspiration yielded a small amount of blood tinged fluid but no pus was obtained

An antero axillary incision was made on the left side, a portion of two ribs was removed and a pocket lined by soft necrotic and fibrinlike tissues was entered It could not be determined whether this was in the lung or between the lobes and, after sizable portions of tissue were removed, the wound was packed wide open with penicillin gauze The pathologic examination revealed nodular gray tissue with a microscopic appearance of great variation Much necrosis was seen along with cellular areas containing fibroblastic proliferation variable sized mononuclear cells lymphocytes, and multinucleated cells Tubular remnants of bronchioles could be seen surrounded by sheets of large mononuclear cells some of which were in mitosis

The interpretation of the slide was that of an epithelial neoplasm although at the time one of us (R J T) thought the lesion was Hodgkin's disease This opinion was partially corroborated at the University of Illinois, where the opinion was expressed that it was Hodgkin's sarcoma (Fig 3)

The patient received x ray therapy and was discharged six weeks after admission The complete clearing of the left lung did not occur until September, 1946 (Fig 2)

The patient has had two children since but at the time of the second pregnancy, she developed nodes in the right cervical area and received x ray therapy Because of recurrence of chest pain and shortness of breath, a therapeutic abortion was performed in 1951 following a third pregnancy At this time, chest x ray revealed a widening of the mediastinal shadow

In July, 1953, fluoroscopy revealed further enlargement of mediastinal widening and nitrogen mustard was given

The last admission was in March, 1954, at which time the patient was markedly icteric, emaciated, and preterminal She died and the autopsy revealed tissue involvement of the retroperitoneal and mediastinal areas, pancreas, spleen, kidney, adrenal glands, and lungs The interpretation was reticulum cell lymphosarcoma but the tissue sections demonstrated pronounced areas of fibrosis with but few scattered areas of cellular infiltration resembling that seen in the surgical specimen These changes resembled the so called "burnt out" Hodgkin's disease (Fig 4)

CASE 2—February, 1947 The patient (A F) a 59 year old white woman, had complained of a mild, dry cough, decreased appetite, and weakness for four to five months Spicy foods had caused "stomach distress" following eating but gastrointestinal x rays were negative The chest roentgenograms revealed a dense, fairly well demarcated mass in the right lower lobe at the cardiophrenic angle

The laboratory data revealed a RBC of 3 7 million, with hemoglobin 11 5 Gm , and WBC, 5 900 The differential count was normal and urine and Wassermann tests were negative Sputum specimens were scant and negative for tubercle bacilli

Bronchoscopic examination revealed compression of the right lower lobe bronchus and aspiration of the posterior basilar segment yielded a small amount of clotted blood

Thoracotomy was done at which time a lemon sized mass was felt in the lower lobe The overlying pleura was slightly puckered and the impression was that the lesion was carcinomatous The lung along with lymph nodes at the hilus was removed [illegible] valescence was uneventful

The pathologic examination revealed a firm mass in the lower lobe which on cut surface had *an irregular, indistinct border of gray color with flecks of carbon pigment* It had occupied slightly less than one-third of the collapsed lower lobe. The lymph nodes were mainly anthracotic.

Microscopically, the sections consisted of numerous small lymphocytes infiltrating the lung parenchyma and obliterating all the architecture with the exception of remnants of bronchi and bronchioles. The lymph nodes were negative.

Fig 1

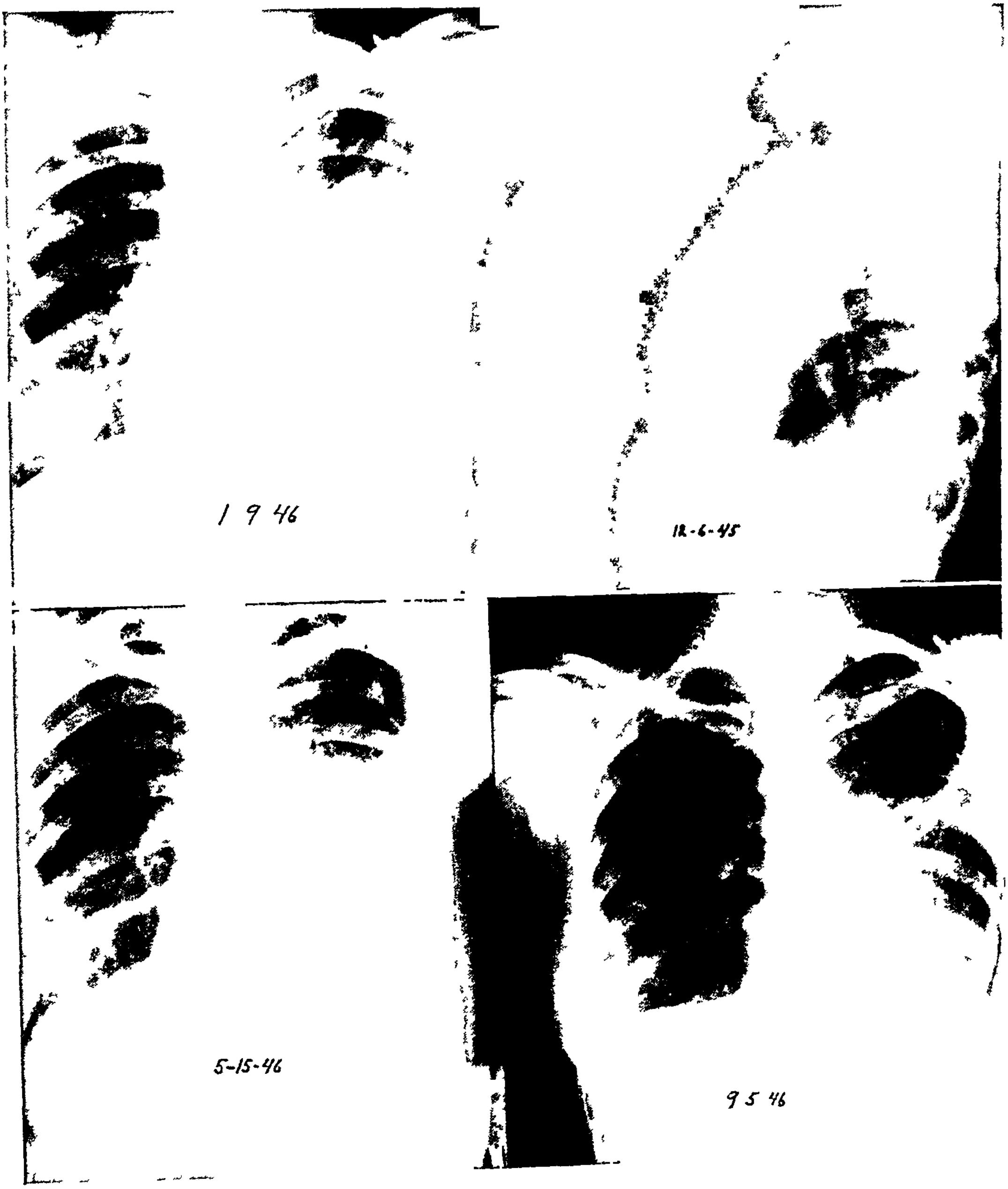


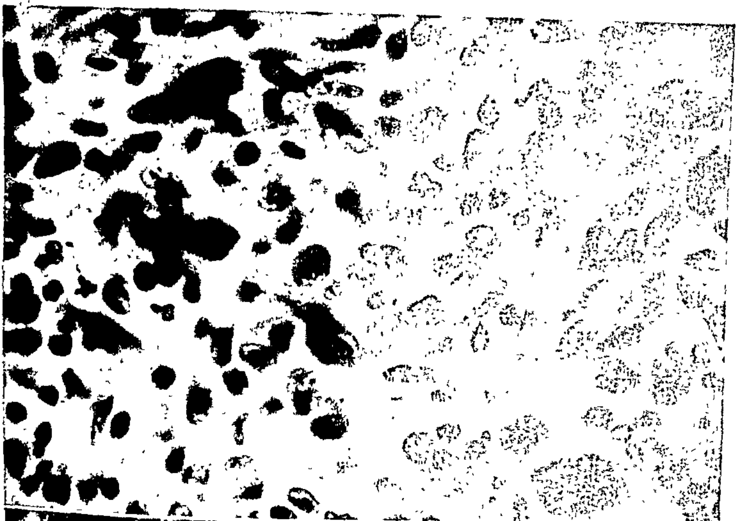
Fig 2.

Fig 1 —Case 1. Mass in left chest, preoperative x-ray
Fig 2 —Case 1. Postoperative chest x-rays Clearing of process in lung required seven months.

The final diagnosis was small-cell lymphosarcoma.

The patient re-entered the hospital three months later because of slight difficulty in breathing and swallowing accompanied by occasional palpitation. She was given a course of nitrogen mustard.

Fig. 3.



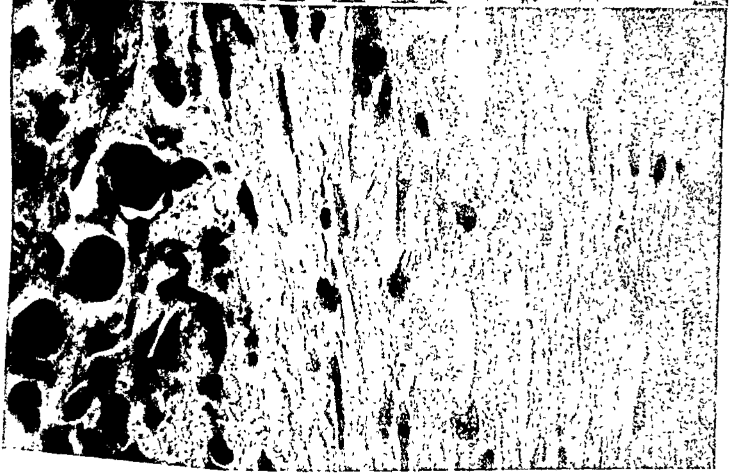


Fig. 4.

Fig. 3.—Case 1. Surgical specimen—pleomorphism and many giant cells—Hodgkin's sarcoma.

Fig. 4.—Case 1. Autopsy specimen—8 years later. Pronounced connective tissue proliferation; degenerative changes in tumor infiltrate.

The patient was not seen again. Death occurred in October, 1950, and the records of the referring doctor stated that it was due to cardiac failure.

CASE 3.—November, 1948. The patient (C. S.) was a 70-year-old white man, who entered the hospital with complaints of shortness of breath which had increased in severity during two years and a cough, of two months' duration, productive of whitish phlegm streaked with blood. He had been hospitalized one week elsewhere because of a diagnosis of pneumonia and was improved but entered St. Luke's Hospital at this time because of the persistent blood-streaked sputum and because of an abnormal shadow seen in the chest x-ray. Two years before the patient had been hospitalized because of a heart attack and since then had had occasional precordial pain on exertion.

The physical examination revealed a somewhat short, stocky, elderly white man in no apparent distress. There were no enlarged lymph nodes in the cervical, axillary, or inguinal regions. The heart and lungs were normal, and the blood pressure was 130/80. There was a left inguinal hernia which could be reduced and a large soft tumor mass in the lumbar region, extending on both left and right sides, which was thought to be a lipoma.

The laboratory studies revealed negative serology and a slight trace of albuminuria. The RBC was 4.2 million; hemoglobin, 12.9 Gm.; and the WBC, 9,300. The nonprotein nitrogen was 44 mg. per cent.

The chest x-ray revealed a solitary density in the left upper lobe just lateral and superior to the hilus. It was not sharply circumscribed, having an irregular border which merged into the adjacent lung tissue. An x-ray two months later revealed no appreciable change.

Bronchoscopy revealed slight compression of the upper lobe and broadening of the spur between the lingula and upper segmental bronchi. Biopsy was taken from the area and secretions aspirated for cytologic study, both of which were negative for tumor. The patient was discharged after the bronchoscopy but readmitted in two months because of the persistence of the hemoptysis. Repeat chest x-rays revealed no appreciable change.

A thoracotomy was done, at which time a firm mass was felt in the left upper lobe. Although it was thought likely to be a carcinoma, only the upper lobe was removed because of the patient's age and previous coronary history. The patient had an uneventful convalescence and was discharged two and one-half weeks postoperatively.

The pathologic examination revealed a mass, measuring 4.5 cm. in greatest diameter and having a gray cut surface with scattered areas of black. Microscopically, the lung tissue was replaced by dense masses of small lymphocytes. These areas were not arranged in follicles and did not have a sinusoid pattern but replaced the parenchyma and surrounded the small bronchi, bronchioles, and vessels which could be identified. The pathologic diagnosis was lymphosarcoma (small-cell) of the lung.

The patient was seen two years later, at which time there was no evidence of recurrence and the chest x-ray revealed good expansion of the remaining lower lobe. He has been lost to follow-up.

CASE 4.—October, 1949. The patient (W. J.), a 53-year-old Negro man, had complained of slight shortness of breath and a cough for the previous two years with a gradual increase in symptoms accompanied by the development of mucoid expectoration in the past four to six weeks. He had had one episode of blood-streaked sputum about a year before. A mobile unit x-ray film taken in September, 1949, revealed an abnormality in the right lung field and admission to Research and Educational Hospital followed.

Physical examination revealed a short, stocky Negro man in no obvious respiratory distress with normal pulse and temperature and blood pressure of 148/80. No enlarged nodes could be palpated in the cervical, axillary, or inguinal areas. Auscultation of the chest revealed dry râles in the right lung posteriorly. X-rays of the chest revealed a density in the right upper lobe which was considered to be an infarction or atypical pneumonitis.

Bronchoscopy was done on two occasions and revealed a compression and stenosis of the right upper lobe orifice.

Thoracotomy was done in November 1949, at which time a firm mass was found in the upper lobe along the major fissure The lobe was smaller in size and on positive pressure no expansion of the area occurred Because of the reported stenosis, the upper lobe was removed The patient had an uneventful convalescence

Pathologic examination revealed a firm mass of [illegible] cm in diameter in the peripheral portion of the lobe The cut surface had a gray pink color with scattered anthracotic patches No point of origin could be identified in the opened bronchi

Microscopically, diffuse infiltration of mature lymphocytic cells was seen throughout the tissues

The final diagnosis was lymphoblastoma of the [illegible] upper lobe, low grade lymphosarcoma form

CASE 5—November, 1949 The patient (I W) was a [illegible]4 year old white woman who became ill in May, 1949, noting "creaking" sounds in the [illegible] when lying down This was followed by the development of a cough which was soon productive of green yellow, foul smelling material She had pain in the right chest, [illegible] attacks with the spasms of coughing, and had lost 32 pounds since the preceding [illegible] An x ray made in 1947 by a mobile unit was reported as negative but one made in July [illegible]49, was abnormal The latter report had resulted in previous hospitalization and bronch [illegible] ic studies (July and September, 1949)

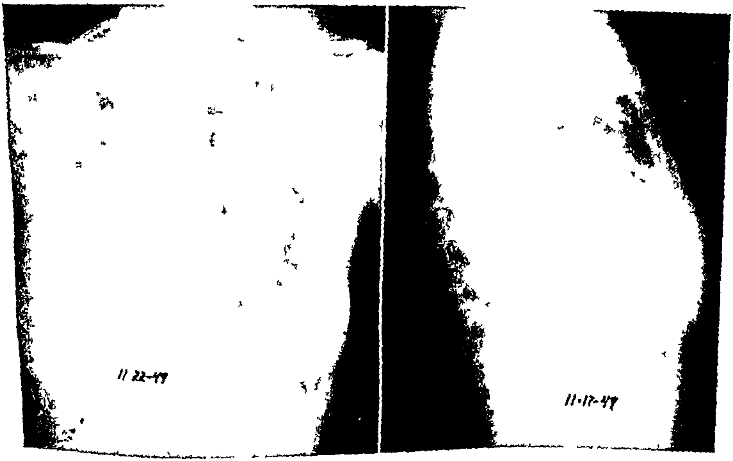


Fig 5—Case 5 Large cavity lesion in upper lobe right lung Lipiodol residue in left lung

On physical examination the patient appeared chronically ill with a temperature of 100° F pulse 90 respiration [illegible]4 and there were slight clubbing and cyanosis of the fingers There were a few small lymph nodes in the neck but no inguinal or axillary adenopathy There was dullness over the entire right chest with many coarse bubbling rales The liver and spleen were not palpable

The laboratory data revealed negative urine and serology The RBC was 3 7 million, hemoglobin, 12 2, and hematocrit 35 5 Sputum and bronchial aspirations were negative [illegible] were negative f[illegible] for tubercle bacilli as was guinea pig inoculation [illegible] on of th[illegible]

Chest x rays revealed a density occupying [illegible] which gradually increased so that the entire [illegible] cavity and fluid level appeared in the upper [illegible] to the right upper lobe (Fig 5)

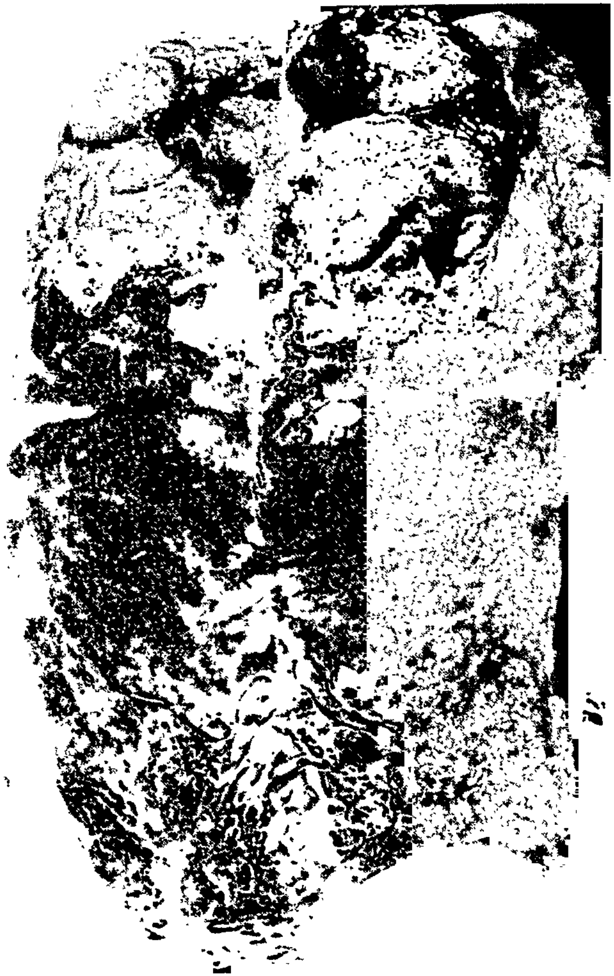


Fig. 6.

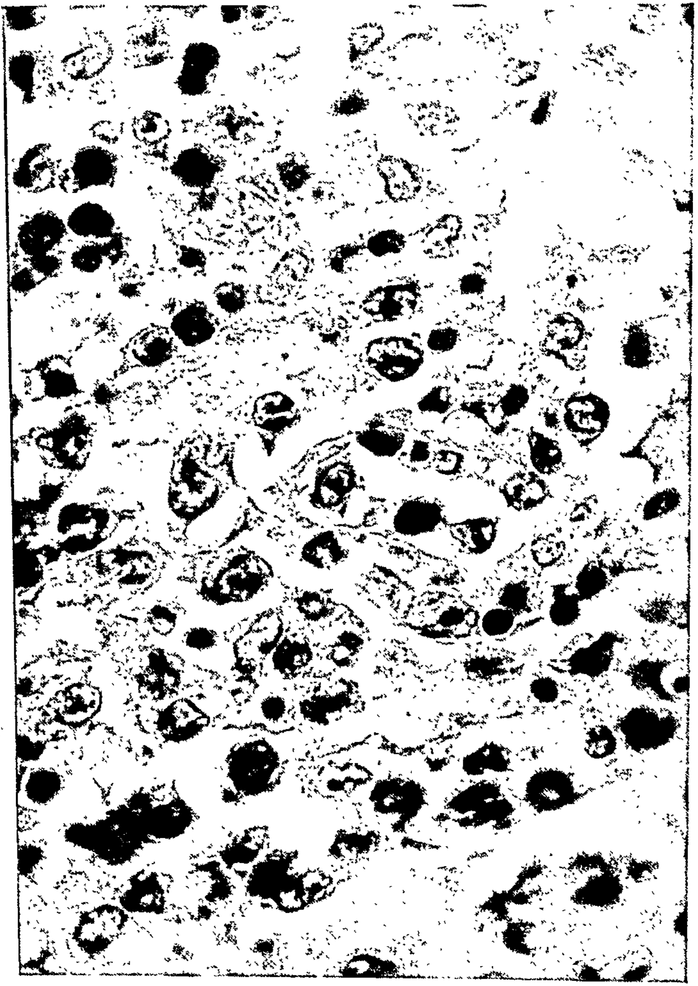


Fig. 7.

Fig. 6.—Case 5. Resected right lung, cut surface. Large cavity with thick walls, upper lobe; consolidation in lower and middle lobes.

Fig. 7.—Case 5. Immature type of lymphoma. Clasmatocytoma or reticulum-cell lymphosarcoma.

The bronchoscopic reports and pathologic interpretations were as follows:

July 28, 1949: A granular, whitish mass was seen at the right side of the carina occluding entrance to the right main bronchus. The bronchoscope could not be passed beyond the mass.

Biopsy revealed fragments of hyaline cartilage and masses of necrotic tissue with ghosts of cellular structures.

Diagnosis: Necrotic tissue, in all likelihood neoplastic.

Sept. 10, 1949: A granular fungating mass was seen to arise from the right side of trachea, 1 cm. above the carina, and it encircled the orifice of the right main bronchus.

Pathology: Bits of squamous and respiratory epithelium were present. Many polys and lymphocytes were present beneath the epithelium. Areas of necrosis were present and suggestive of caseation. Necrosis was present but no acid-fast bacilli were seen.

Diagnosis: Necrosis with acute and chronic inflammation and squamous-cell metaplasia.

Oct. 29, 1949: A reddish mass partially obstructed the right main bronchus. A biopsy was taken.

Pathology: Dense connective tissue and granulation tissue were present.

Diagnosis: Reactive changes not typically inflammatory. Larger cells present and a peculiar alignment were suggestive of a lymphomatous process.

Nov. 18, 1949: Two raised areas were present 2 cm. above the carina on the right lateral wall of the trachea. The mucosa was smooth. There was a stenosis of the right upper lobe but tissue was removed from within the orifice of the upper lobe. The carina was widened.

Pathology: Hemorrhagic loose areolar and dense collagenous tissues were infiltrated with many acute and chronic inflammatory cells.

Diagnosis: Acute and chronic inflammation of the bronchus.

A thoracotomy was done on Nov. 29, 1949. Many adhesions were encountered. The entire upper lobe was firm with a fluctuant central area. The entire lung was removed with closure of the bronchus at the carina (Fig. 6).

The patient made an uneventful recovery and on the second week postoperatively was given a course of nitrogen mustard therapy at the end of which time she was discharged from the hospital.

The pathologic examination revealed a large cavity 10 cm. in diameter in the upper lobe. The lining of this consisted of ragged, ulcerated, tan and gray tissues. There was a defect at the hilus, which communicated directly with the cavity, the wall of which was as large as 3 to 4 cm. in thickness and resembled "fish flesh" in appearance.

The middle and lower lobes were adherent by dense adhesions and on cut surface the bronchi and bronchioles contained creamy exudate. The nodes at the hilus were enlarged and firm.

Histologically, much of the lung tissue was necrotic, pink, amorphous material. There were *many* scattered areas consisting of very pleomorphic large and small cells invading the atelectatic parenchyma. Some were multinucleated and giant in size. The lymph node contained many inflammatory cells.

Diagnosis: Lymphoblastoma of right upper lobe with extensive necrosis; acute bronchitis and chronic bronchopneumonia; and hyperplasia of hilar nodes (Fig. 7).

This patient is alive and well at present, almost five and one-half years since surgery and has had three children since then.

Case 6.—October, 1949. The patient (M. S.), was a 25-year-old white woman, a housewife, who entered St. Luke's Hospital with a history of generalized itching, swelling of the right side of the neck and a weight loss of 10 to 12 pounds during the previous six months. Following x-ray studies of the chest in August, the patient had entered a tuberculosis sanatorium where streptomycin therapy was given for eight weeks' time (Fig. 8). Despite treatment, there was a progressive increase in the number and size of the nodules and she was transferred for further work-up.

The physical examination revealed a young white woman with numerous areas of excoriation from scratching over most of the body surface.

There were several 2 to 3 cm. firm nodes palpable in the right cervical region and a few pea-sized nodes on the left side. The lungs and heart were essentially normal with the blood pressure of 135/70. The abdomen was negative.

The laboratory data revealed negative urine and negative serology with a RBC of 3.46 million, a WBC of 16,400, and hemoglobin of 10.3 Gm.

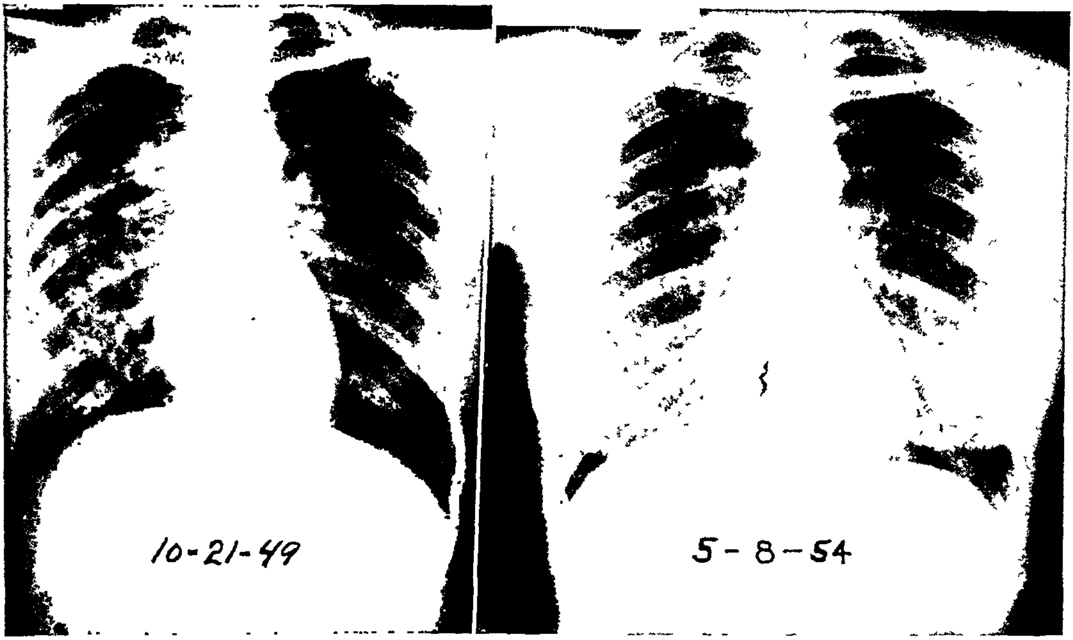


Fig. 8.—Case 6. Nodular dissemination of Hodgkin's disease. First interpretation was pulmonary tuberculosis.

There were finely mottled, soft densities scattered throughout the right lung field with only faint suggestion of left lung involvement. An x-ray in October revealed coalescence, enlargement, and increase in these areas. A chest film in 1954 revealed almost complete disappearance of the pulmonary infiltrations.

A cervical node on the right side was removed, and the histologic diagnosis was Hodgkin's disease. The patient received nitrogen mustard for four consecutive days.

The second admission was in February, 1952, because of edema of the left lower extremity and lymph node enlargement in the inguinal area. She received another series of nitrogen mustard treatments on four consecutive days.

The patient has not been seen since 1952 but she is still alive. Personal communication with the family doctor revealed that the patient has had several repeated series of both x-ray and mustard treatments.

Case 7.—December, 1953. The patient (G. C.) was a 46-year-old white woman who had had an abnormality discovered in an x-ray of her chest made by a Mobile Survey Unit, six weeks before entrance to St. Luke's Hospital. She had had previous films made which had been reported as negative. She was essentially without complaints except for slight fatigue.

The physical examination failed to reveal any essential abnormality. No palpable nodes were present in the neck, axillary, or inguinal area. The heart and lungs were normal. Blood pressure was 120/80. Liver and spleen were not palpable.

The laboratory findings revealed negative urine and serology The RBC was 4 8 million, WBC, 8,300, hemoglobin, 12 6, with lymphocytes 6, monocytes 4, neutrophils 83, and bands 7

The chest x rays are shown in Fig 9

Bronchoscopic examination revealed slight injection of the mucosa in the left lower lobe bronchus but no masses or compression of the segmental orifices Aspiration deep in the lower lobe bronchus caused pronounced oozing from the bronchus and this material was sent for Papanicolaou and cell block studies, both of which were negative

At thoracotomy, a firm mass was found in the left lower lobe and on the interlobar surface the pleura had a brown discoloration, suggesting an infarction Several small nodes were present along the bronchus and these were removed with the entire lower lobe

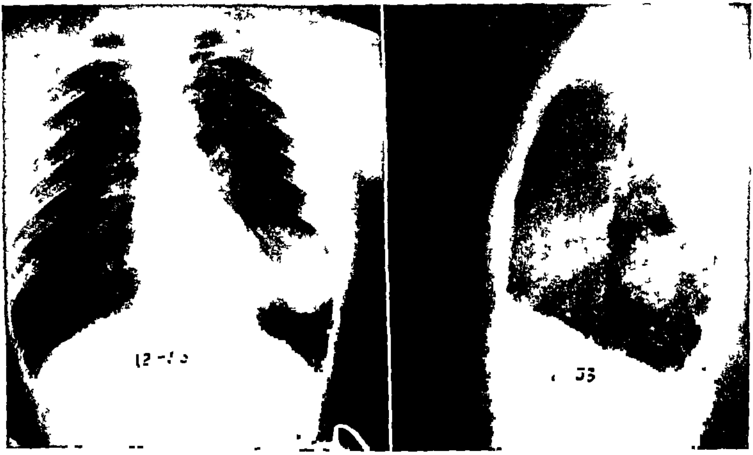


Fig 9—Case 7 Solitary mass in lower lobe of left lung

The pathologic examination revealed a mass which measured 7 cm in greatest diameter on cut surface and had a grayish tan color (Fig 10) No distinct point of bronchial origin was found Histologically, the mass consisted of many small lymphocytes without distinct sinusoids or cortical nodules These surrounded bronchi, bronchioles, and small vessels and contained scattered carbon deposits The cells replaced the alveolar lung parenchyma and the pathologic diagnosis was small cell lymphosarcoma (Fig 11)

The resected lymph nodes were negative The convalescence was uneventful, and the patient is alive and well almost eighteen months following surgery

CASE 8—July, 1942 The patient (C D) was a 48 year old white man who entered St Luke's Hospital in July, 1942, for bronchoscopic study and evaluation of an abnormality in a chest x ray taken when he attempted to obtain a commission in the Armed Forces Other than occasional cough and slight expectoration associated with colds, he felt that he was entirely well

The physical examination was entirely within normal limits except for slight generalized obesity The laboratory data were within normal limits and bronchial secretions were negative for tubercle bacilli

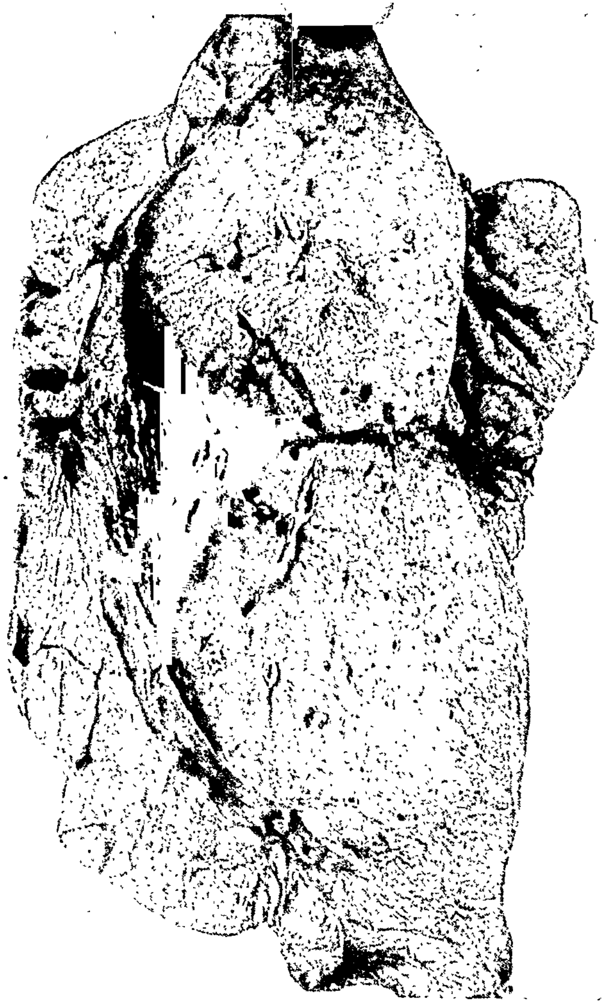


Fig. 10.

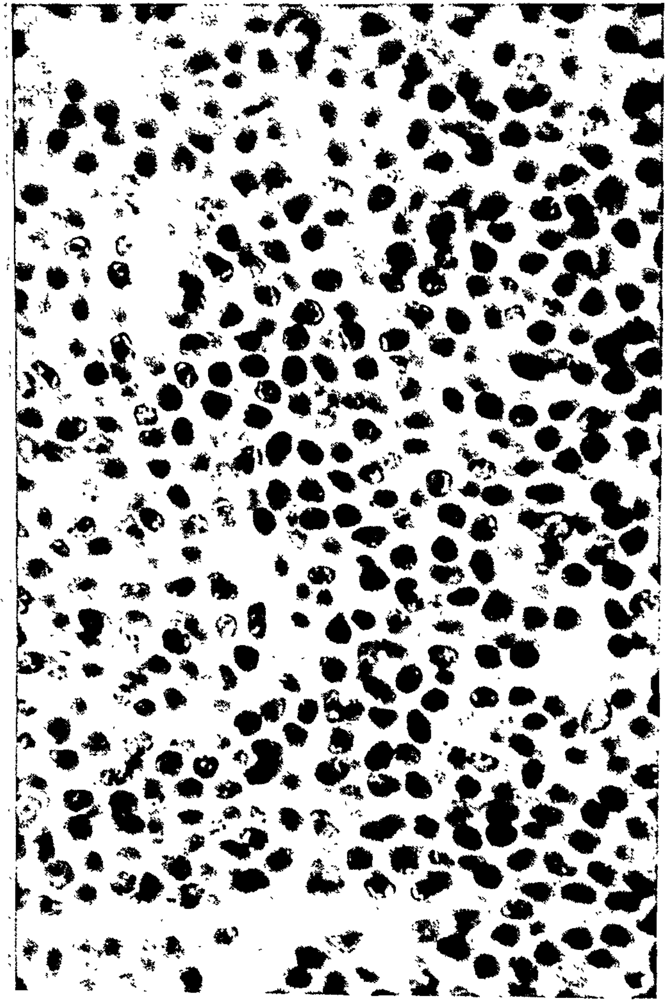


Fig. 11.

Fig. 10.—Case 7. Cut surface of lower lobe. The tumor was homogeneous, bulging and had fine irregular margin.

Fig. 11.—Case 7. Lymphocytic lymphoma. The majority of cells are fairly mature; occasional lymphoblasts present.

The chest x rays revealed a fusiform density at the lateral chest wall opposite the fourth through sixth ribs laterally and a soft nodular ill defined density opposite the left pulmonary hilus

Bronchoscopic studies were essentially normal

The second admission was in October, 1947, at which time the patient complained of slight shortness of breath The chest x rays (Fig 12) revealed enlargement of the density on the left side a new density in the right lung extending from the first to third ribs anteriorly and obliteration of the lower half of the right lung field, suggesting fluid Thoracentesis was done and 2,300 c c of slightly turbid, yellow fluid was obtained Histologically, this was reported as pleural exudate but the majority of the cells were lymphocytes

The third admission was in July, 1948, at which time the patient had marked dyspnea and a temperature of 104° F During the past months, fluid had been removed at monthly intervals totalling about 6 L Since the last aspiration, he had developed fever and pronounced dyspnea Twelve hundred cubic centimeters of slightly turbid fluid were removed from which hemolytic *Staphylococcus aureus* was cultured

The chest x rays at this time were essentially the same as of October, 1947 (Fig 13), except for an increase in the size of the lesions in both chest cavities and the appearance of a third lesion in the costophrenic angle on the right side above the fluid level

A right thoracotomy was done in August, 1948 The two larger areas were extrapleural in location and consisted of "soft fatlike tumor" with minute cystic spaces A frozen section of this was reported as small cell or round cell carcinoma The lower mass consisted of fibrin and extended between the lobes The remaining pleura over the lower and middle lobes had the appearance of a pyogenic membrane and restricted the complete examination of the lung The extrapleural masses were resected, the fibrin and exudate from the pleural space was removed, and decortication of the lower and middle lobes was done The chest was closed with intercostal drainage

The tissue submitted for pathologic examination consisted of several portions, the largest measuring 8 by 7 by 2 5 cm It had a soft consistency with a gray and tan brown cut surface Histologically, the tissue was composed of lymphocyte like cells, having very little cytoplasm and a vesicular nucleus Very little stroma and only an occasional germinal center were present (Fig 14)

The hospital course was prolonged because of the persistence of the pleural space infection, and the patient was finally discharged with a residual pocket of about 90 c c in size During the hospitalization, he received roentgen therapy over the right thorax following which there was complete disappearance of the lesion in the left chest as well

The fourth hospital admission was in November, 1948, at which time the pleural pocket was unroofed removing a portion of two ribs Pathologic examination of the thickened parietal pleura did not reveal any evidence of lymphosarcoma at that time

In November, 1953, the patient developed diarrhea and blood in the stools, and on proctoscopic examination an adenocarcinoma was found at the 12 cm level on the anterior aspect of the rectum An abdominoperineal resection was performed by Dr Pontius and was followed by an uneventful convalescence

In February, 1954, the patient had an episode of blood streaked sputum An x ray (Fig 15) revealed the rib defects from the open drainage procedure on the right side and an ill defined mass off the left hilus in the area of the previous mass in the left lung

Bronchus examination revealed the bleeding source to be in the right lower lobe The left side was entirely normal The pathologic study revealed only hemorrhagic bronchial fluid

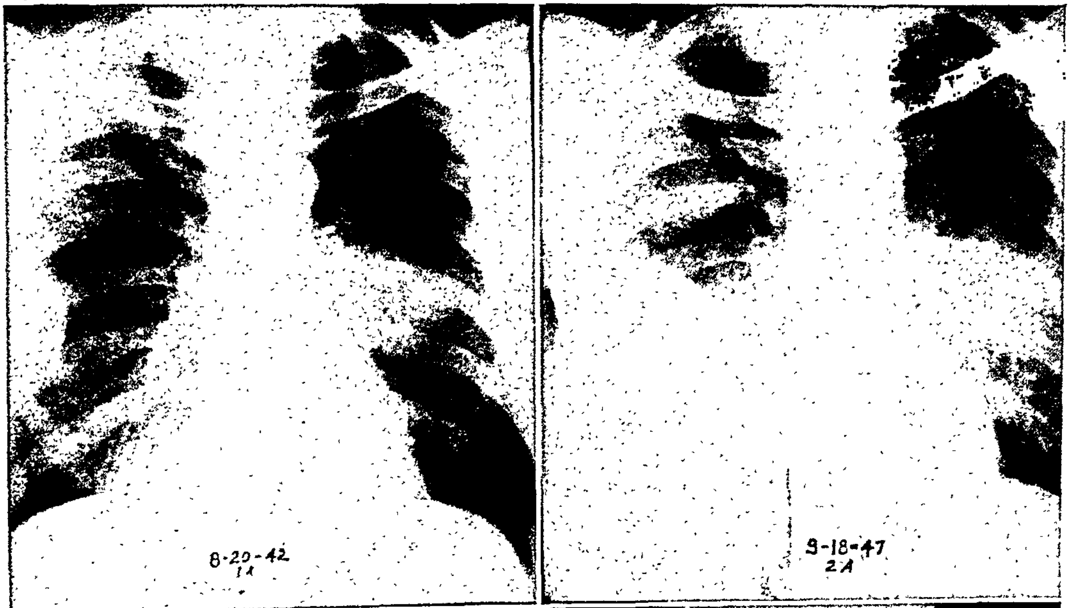
Roentgen therapy was again given over the left chest and the last x ray, March, 1954 (Fig 15), demonstrated disappearance of the mass in the left lung Bronchoscopy was repeated in March because of the persistence of the blood streaked sputum and again confirmed the site of the bleeding in the lateral basilar bronchus of the right lower lobe Cell block and Papanicolaou examinations were negative for tumor

The bleeding disappeared after antibiotic therapy and since March, 1954, there has been no recurrence

The patient is alive and well at the present time, thirteen years after the first known evidence of lymphosarcoma, having undergone surgical and roentgen therapy for it, plus a cholecystectomy at another hospital and an abdominoperineal resection for adenocarcinoma of the rectum in that time.

Case 9.—December, 1947. The patient (E. H.) was a 43-year-old white woman who had had pneumonia five months previously and had been treated at another hospital with

Fig. 12.



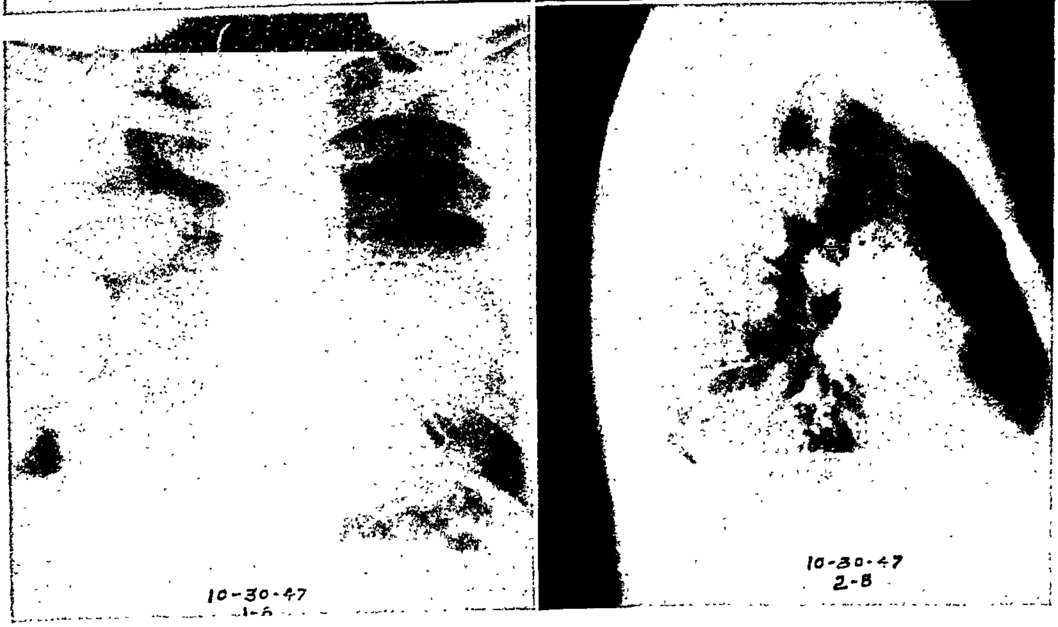


Fig. 13.

Fig. 12.—Case 8. Enlargement of pulmonary shadows with pleural fluid; five-year interval.

Fig. 13.—Case 8. Postthoracentesis. Mass on left is in left lung; nodules on right are extrapleural.

antibiotic medication Following this, she had a persistent cough, expectorated thick, brownish material, and had episodes of fever She was "anemic" for which liver medication was given She developed hemoptysis and was rehospitalized where x ray studies of the chest suggested fluid in the left pleural cavity She was admitted to St Luke's Hospital in December for bronchoscopic study and evaluation

Physical examination revealed changes in the left chest suggestive of fluid and there were enlarged lymph nodes present in the right cervical and right inguinal regions

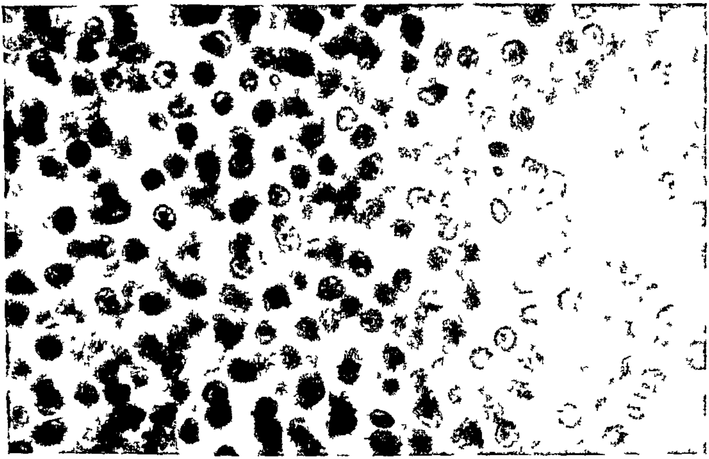


Fig 14—Case 8 Lymphocytic lymphoma Cells are immature lymphocytes

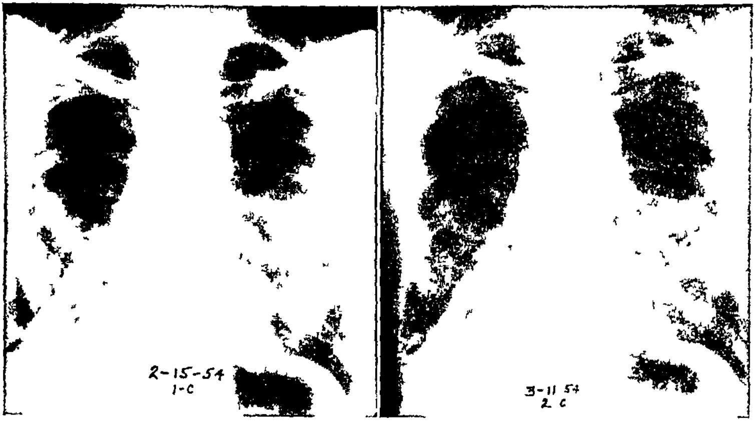


Fig 15—Case 8 Recurrence of mass in left lung and clearing following x ray irradiation

The laboratory data revealed RBC, 3.8 million; WBC, 8,100; and hemoglobin, 11.5 Gm. The urine and serology were negative, and the sputum was negative for tubercle bacilli.

The x-ray studies demonstrated the left pleural cavity to be obliterated except for a small area of aerated lung at the apex.

The bronchoscopic examination revealed a broadened carina and compression of the left main bronchus. Stenosis was present at the upper lobe orifice and the mucosa was thickened and friable. Biopsy was obtained from this area and the report was questionable lymphosarcoma of the bronchial mucosa.

Fluid was aspirated from the chest and, histologically, showed numerous lymphocytes.

A biopsy of an inguinal node was done and the histologic report was small-cell lymphosarcoma.

She re-entered the hospital in July, 1949, because of swelling of the lower extremities. Physical examination revealed swelling of both lower extremities, more marked on the right, and a recurrence of nodes in the right inguinal area. The patient received x-ray therapy. Chest x-ray revealed no recurrence of fluid or widening of the mediastinum.

She was readmitted in December, 1949, because of persistence of swelling in the right thigh. At this time, nitrogen mustard was given and improvement in the swelling occurred.

In February, 1951, she was readmitted because of back pain and swelling in the neck. Treatment consisted of x-ray therapy to the neck and lumbar spine and there was improvement but the cervical nodal enlargement recurred and more x-ray was given in May, 1951.

Her last admission was in June, 1951, when she was preterminal. Mustard therapy was given but no improvement followed. The patient died in July, 1951, slightly less than four years after the onset of the disease.

Review of lymph node biopsy revealed a very abnormal cellular pattern. The cells were variable in size and shape and scattered eosinophils were present. A very occasional, large, double nucleated cell was seen suggesting Sternberg-Reed-cells. Necrosis was not apparent. The diagnosis was finally made of early Hodgkin's disease without necrosis or fibrosis.

CASE 10.—December, 1949. The patient (I. E.) was a 40-year-old white woman who had developed pleurisy on the right side six months before and two months previously had noticed difficulty in breathing. She had been treated elsewhere and clear or slightly blood-tinged fluid had been aspirated from the left chest several times.

On physical examination, no enlarged nodes were palpable but dullness to percussion and absent breath sounds were the abnormal findings on the left side.

Laboratory studies revealed negative urine and serology. RBC was 4.1 million; WBC, 8,300; and hemoglobin, 12.9 Gm. The differential count was lymphocytes 6, monocytes 4, neutrophils 75, basophils 2, eosinophils 8, bands 5. The aspirated fluid was negative to smear and culture.

Treatment consisted of thoracentesis on two occasions, removing 1,500 c.c. of clear, straw-colored fluid and, two weeks later, 2,400 c.c. of light brown fluid. Between these two aspirations, a course of nitrogen mustard was given.

Pathologically, the fluid contained numerous lymphocytes and the diagnosis of the blocked centrifuged specimen was of marked lymphocytic infiltration of pleural fluid.

The patient re-entered the hospital a year later because of recurrence of fluid and 1 L. was again removed. The histologic report again was of marked lymphocytic infiltration of pleural fluid, and the patient received another course of nitrogen mustard.

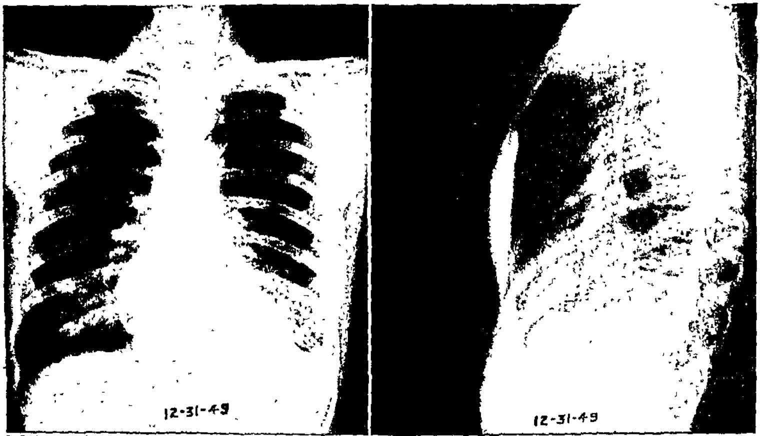
In 1952, following aspiration of recurrent fluid, the patient developed a hemolytic *Staphylococcus aureus* empyema, which was treated by aspiration and antibiotic therapy. Surgical drainage was not done because of the associated lymphosarcoma and the patient was discharged with a residual density in the left lower chest. She received x-ray therapy over the left chest (Figs. 16 and 17).

In October, 1954, the patient was admitted because of persistent fever and pain. Needle aspiration revealed thick pus and thoracotomy drainage was accomplished under local anesthesia.

The thickened parietal pleura was submitted for histologic examination and revealed a dense layer of lymphocytic infiltration on one side of the collagenous tissue with a pyogenic layer on the opposite side (Fig. 18). Pathologic diagnosis was lymphosarcoma of the pleura.

The patient was hospitalized two more times in 1955 for nitrogen mustard therapy and x-ray therapy but she died in February, 1955, five and one-half years after the onset of her symptoms. Permission for an autopsy was denied.

Fig. 16.



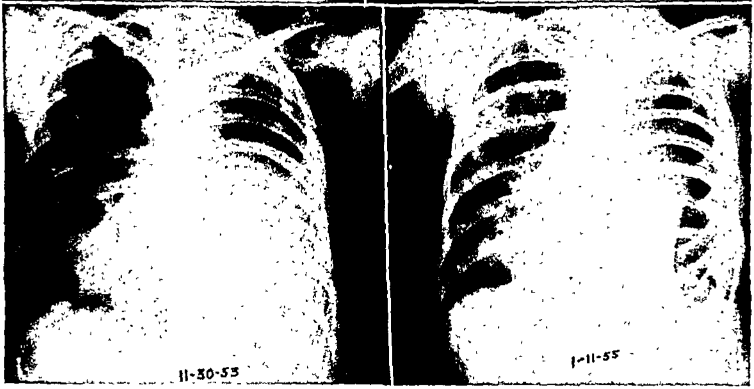


Fig. 17.

Fig. 16.—Case 10. Postthoracentesis x-rays of chest.

Fig. 17.—Case 10. Residual empyema pocket, left chest and postthoracotomy film; spread of lymphoma to right lung.

CASE 11.—October, 1953. The patient (T. M.) was a 67-year-old white woman who had noticed weakness and slight shortness of breath and had been treated elsewhere for heart disease. Because of the increase in her dyspnea, examination and x-rays of the chest by another physician suggested fluid in the right pleural cavity. She entered St. Luke's Hospital, where the essential physical findings were limited to the right pleural cavity and suggested the presence of fluid. The heart was shifted to the left and had a normal sinus rhythm, and the blood pressure was 170/94. The liver was enlarged four fingerbreadths below the costal margin.

The laboratory studies revealed a normal urine and serology. The RBC was 4.5 million; WBC, 4,900; and hemoglobin, 13.2 Gm.

Chest x-rays revealed a density in the right chest, suggesting fluid. Aspiration of the chest was done on two occasions, and 2,400 and 1,600 c.c. of slightly opalescent, straw-colored fluid were obtained.

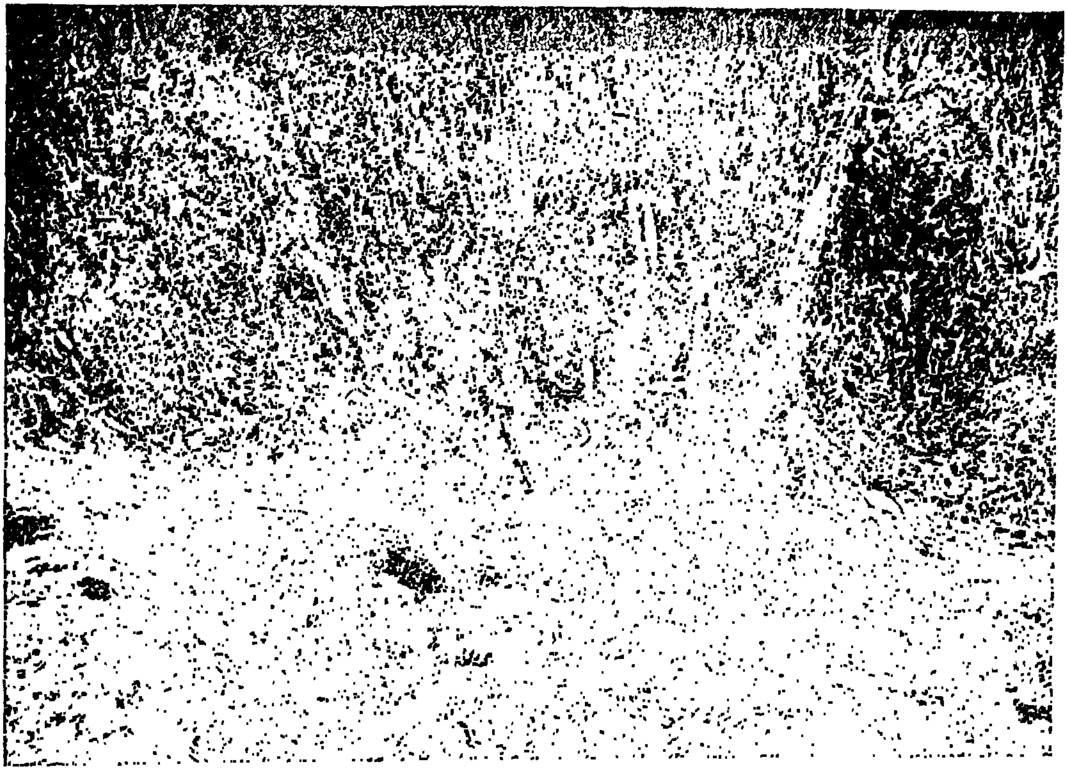


Fig. 18.—Case 10. Hyalinized connective tissue of pleura with dense infiltration of lymphosarcoma above.

The histologic study revealed many lymphocytes, and the diagnosis was submitted as lymphocytic infiltration of pleural fluid. The second fluid specimen revealed similar lymphocytes but to a lesser degree.

Bronchoscopic examination revealed minimal increased redness of the mucosa but no evidence of bronchial compression or changes of the coryna. The aspirated secretions were negative for carcinoma (Papanicolaou and cell block study).

A thoracotomy was done and 1,500 c.c. of fluid removed. There was slight injection of the parietal pleura but no nodular masses were present on the pleura. There were confluent, firm nodes below the right main bronchus extending downward into the pulmonary ligament. Two nodes were present above the azygos vein and a walnut-sized mass was present above the diaphragm. Several of the nodes and the mass above the diaphragm were removed, and on histologic examination were reported as either reticulum-cell lymphosarcoma or Hodgkin's disease.

The patient was given nitrogen mustard after a suitable period of convalescence

Readmission was six months later (June, 1954) She had received x ray therapy for enlarged nodes in neck, axilla, and inguinal area She also had developed pain in the left leg and hip and required a crutch for walking She was given another course of nitrogen mustard in June and in October, 1954 She died in February, 1955, at home, and permission for an autopsy was not obtained

The final reclassification of the node pathology was Hodgkin's disease

CASE 12—November, 1953 The patient (A D) was a 70 year old white woman who had complained of a gradual increase in shortness of breath and a nonproductive cough for the preceding four months She was treated with digitalis and diuretics by her family doctor but her complaints increased After x ray studies revealed fluid in the right pleural cavity, thoracentesis was done which yielded 500 c c of fluid on one occasion and three weeks later, 1 L of slightly blood tinged fluid was aspirated The patient had had acute rheumatic fever as a child of 13

The physical findings were essentially confined to the right chest, where changes indicative of pleural fluid were elicited The heart was normal in rhythm and rate with a blood pressure of 138/80 A few small nodes were palpable in each axilla

The laboratory findings revealed a RBC of 4 9 million, WBC, 10,400, and hemoglobin, 14 3 Gm The differential count was lymphocytes 24, monocytes 3, polymorphonuclear leukocytes 60, eosinophils 4, and bands 9

X rays of the chest revealed obliteration of the lung markings by fluid

Approximately 1 500 c c of amber fluid were removed from the right pleural cavity Following aspiration, a fusiform widening of the right side of the mediastinum was seen Histologic examination of the blocked centrifuged specimen revealed myriads of lymphocytes and the suggestion of the pathologist was that the patient might have a lymphoma Three small nodes were removed from the axilla and were negative histologically She was given nitrogen mustard for four consecutive days and a chest x ray subsequently revealed narrowing of the mediastinum and only a small amount of fluid in the pleural cavity

She was readmitted five weeks later complaining of shortness of breath and swelling of the ankles Fluid was aspirated from the chest twice, six and twelve days later

Two liters of fluid was removed which had the same histologic findings Chest x ray revealed marked mediastinal widening and hydropneumothorax Following this, roentgen therapy was given

She was readmitted six weeks later with complaints of shortness of breath, anorexia, weight loss, and diplopia The chest contained fluid, the liver and spleen were enlarged, nodes were present in the neck and there was a lateral deviation of the right eye The right chest was aspirated and 2 200 c c of fluid was obtained Nitrogen mustard therapy was repeated and the patient was discharged with questionable improvement

Death occurred in April, 1954 at home and permission for autopsy was not obtained

DISCUSSION

Analysis of this group revealed that the symptomatology was absent, minimal, or pronounced At least 3 patients had silent pulmonary shadows, 1 had occasional blood tinged expectoration, and 2 had overwhelming symptoms of toxemia with anemia, fever, severe cough with varying degrees of productivity, and marked weight loss As would be expected, shortness of breath was the chief complaint of those in the pleural fluid group The age group varied from 24 to 70 years but there was a definite tendency toward a young age incidence Two of the patients, who were in the third decade, had Hodgkin's disease which has a tendency toward early occurrence in life (Gall and Mallory[3])

The sex incidence was in contrast to that in the collected series of Beck and Reganis[8] Seventy five per cent of the patients were women

The roentgenologic changes similarly were extremely variable although a circumscribed density was present in 4 patients. One of the patients with pleural fluid had two densities which were extrapleural on the same side and a density in the contralateral lung. Two patients had massive pulmonary involvement, in 1 of whom bronchial occlusion, consolidation, and abscess formation were present. A disseminated nodular disease present in both lungs but primarily on the right side was seen in 1 patient in this group. This corresponded to the Class II type reported in Robbin's roentgenographic study, which group incidentally comprised over 85 per cent of the reported cases.

The patients with pleural fluid all exhibited typical fluid shadows, and in 1, mediastinal changes subsequently occurred.

Examples of the variations in roentgenologic appearance accompany the briefs of the case histories.

Likewise, the bronchoscopic findings are listed in the abstracted histories. For the most part, information obtained was limited. One patient was bronchoscoped four times prior to resection and with biopsy material obtained on one of these occasions changes suggesting a lymphoma were reported. One patient in the pleural fluid group had a biopsy of the wall of the left main bronchus, the site of compression at the left upper lobe orifice, and this revealed lymphocytic infiltration of the bronchial mucosa, possibly lymphosarcoma. These two instances were the only examples in the series in which diagnosis was suggested by bronchial material. In the remainder, negative reports or instances of varying degrees of bronchial compression were described.

Of particular interest were the pathologic findings. Four patients in the resected group had a solitary mass which varied from a few centimeters up to 8 to 10 cm. in greatest diameter. These were firm, rubbery in consistency, and had a bulging cut surface varying from pinkish-gray to tan-gray color. The margins in most instances were finely irregular and what might be described as fuzzy, blending into the surrounding parenchyma. The tumor mass could not be enucleated from the lung; however, the masses of tumor which were resected in Case 8 were separated from the extrapleural tissues by a fairly distinct line of cleavage. This tumor tissue also had a gray and tan cut surface.

One resected specimen contained a large abscess cavity and almost the entire upper lobe was involved. Again the cut surfaces had a pink-gray color and the pathologist chose the term "fish flesh" in describing it.

The most common histologic type was the lymphocytic lymphosarcoma. Microscopically, there was a diffuse infiltration of small lymphocytic cells without semblance of normal lymphoid architecture. Sinusoids, trabeculae and germinal centers were totally lacking but remnants of small bronchi, bronchioles, and vessels could be identified. There were occasional less differentiated cells which appeared as larger cells with a more vesicular, less heavily stained nucleus and occasional nucleolus, which could be interpreted as lymphoblasts.

The solitary tumor lesions in 4 patients were of this type. In one patient with extrapleural masses and pleural fluid, the tumor pathology in the contralateral lung also was of this type and Case 10, a patient with recurring pleural fluid, demonstrated the identical histologic changes in the thickened scar tissue excised from the parietal pleura The photomicrographs in Figs 11 and 15 are of this histologic type

The histologic picture of the lung resected in Case 5 was that of a very immature type cell, large, variable in size and shape and in some instances having cytoplasmic processes extending from the cells (Fig 7) The nucleus contained scant chromatin and frequently nucleoli were apparent Considerable necrosis with a variegated assortment of scattered polymorphonuclear and mononuclear inflammatory cells were also seen

Although this was classified as a lymphoblastoma, it would be more in keeping histologically with a clasmatocytoma (Gall and Mallory[3]) or a reticulum cell sarcoma (Jackson and Parker[17]), depending upon the classification followed It is of interest that in view of the very undifferentiated and immature character of the lesion, this patient is still alive and well without evidence of recurrence, five and one half years following resection

The majority of the reported cases in the literature would seem to fall in the lymphocytic cell type judging from what can be learned from the photomicrographs and cytologic descriptions However, the cases reported by Maier,[6] Spatt and Grayzel, and Grimes, Wenrich, and Stephens[11] are more suggestive of lymphoblastoma types

Case 1, in which only partial resection was done, was histologically the most bizarre and anaplastic lesion of the series The sections were exceedingly pleomorphic with odd sized and shaped cells and numerous multinucleated giant cells Frequent mitoses were evident as well as moderate polymorphonuclear cells especially eosinophils Necrosis and scattered fibrillar areas could be seen Although the first diagnosis was undifferentiated epithelial cancer, review of this material resulted in its classification as Hodgkin's sarcoma Figs 3 and 4 are sections from this patient, the latter specimen being from the autopsy eight and one half years following thoracotomy At this time pronounced dense collagenous tissue was apparent but still discernible were islets of bizarre pleomorphic cells some of which were very dark staining, indicating beginning necrotic changes The appearance of the latter section was very similar to the long standing chronic "burnt out" Hodgkin's disease The long duration of this patient's illness is not in keeping with the generally known prognosis of the Hodgkin's sarcoma group

Last the pathologic features of the pleural fluid group were of interest in that cytologic examination of the blocked centrifuged fluid contained myriads of rather mature lymphocytes Material obtained for histologic study in 2 of these patients at the time of thoracotomy was histologically identical with the lymphocytic type lymphosarcoma described previously In one of these patients the procedure was done to drain an empyema pocket and a section of the thickened pleural tissues revealed a pyogenic cellular reaction on one surface of the dense hyalinized tissue and on the other a very dense, cellular lymphosarcomatous infiltration (Fig 18)

Two patients in this group had lymphocytic infiltration into the pleural fluid and had a final diagnosis of Hodgkin's disease In 1, thoracotomy was done and revealed slightly enlarged lymph nodes in the inferior pulmonary ligament and a single node superior to the azygos vein. Histologically, these were consistent with Hodgkin's disease, demonstrating pleomorphic cellular aggregates, eosinophilic polymorphonuclear leukocytes, and Sternberg-Reed cells. Although the other case did not have thoracotomy, the diagnosis of Hodgkin's disease was made from inguinal node biopsy. Very interestingly, this patient also had the typical lymphosarcoma infiltration which showed in a bronchoscopic biopsy. It is difficult to explain this, as the constancy of the lymphoma lesions is well shown in the figures of Gall and Mallory. About the only change occurring in individual cases was in degree of differentiation and that in only some 23 per cent. It might be possible to explain the bronchial infiltration as a very early stage in which the lymphocytic infiltration was so marked as to obscure the other cellular components.

In 5 patients with parenchymal disease, lobectomy was performed in 3 and pneumonectomy in 2, accomplishing complete removal of the lesion in all 5 cases. The hilar lymph nodes removed along with the lobe or lung were all negative for tumor. One other patient in this group had only partial removal of the tumor because of the widespread involvement and was treated with x-ray and nitrogen mustard therapy (Hodgkin's sarcoma, Case 1). Of interest was the delay in the clearing of the left lung field following irradiation.

The other patient with partial resection was in the pleural fluid group. The surgery consisted of resection of the extrapleural masses in the right chest and decortication of the lower lobe for the complicating superimposed empyema. After the diagnosis was established, roentgen therapy was given over the right chest and the lesion in the left lung disappeared.

The treatment in the pleural fluid group consisted of aspiration, x-ray, and nitrogen mustard therapy. In 2 patients, drainage procedures were necessary because of complicating pyogenic infections and, in 1, biopsy material was obtained to substantiate the diagnosis of lymphoma. One patient had a peripheral node biopsy made for diagnosis, which was also done in 1 patient in the parenchymal group (Case 6).

The results obtained in this series are summarized in Tables I, II, and III, the latter consisting of the resected cases. Three of the 5 patients with total resection are alive, the length of time varying from one and one-third years up to five and one-half years. One has been lost to follow-up, and one survived three years before dying of cardiac failure.

Of the 2 cases with partial resection, 1 survived for more than eight years and the other is alive more than thirteen years from the date of the first x-rays revealing an abnormality, and almost seven years from the time the diagnosis was established by surgical exploration and removal of tumor.

Four of the patients with pleural fluid died in periods ranging from five months to five and one-half years. The patient with the shortest survival time (Case 12) died away from the hospital and autopsy material was not available. This was the only patient in the entire group in whom the only material obtained for the diagnosis was pleural fluid.

TABLE III. RESULTS OF RESECTION IN PULMONARY LYMPHOMA

PATIENT	SITE	HISTOLOGIC DIAGNOSIS	TREATMENT	RESULTS
M. W. 9 45	Left lung	Hodgkin's sarcoma	Subtotal resection, nitrogen mustard, roentgen	Dead, 8½ yr survival
A F 3 47	Right lung	Small cell lymphosarcoma	Right pneumonectomy	Dead, 3½ yr survival
C S 1 49	Left lung	Small cell lymphosarcoma	Left upper lobectomy	Lost to follow up after 2 yr
W J. 11 49	Right lung	Small cell lymphosarcoma	Right upper lobectomy	Alive 5½ yr
I W. 11 49	Right lung	Lymphoblastoma	Right pneumonectomy	Alive 5½ yr.
G C. 12 53	Left lung	Small cell lymphosarcoma	Left lower lobectomy	Alive 1½ yr
C. D 1912	Bilateral	Small cell lymphosarcoma	Subtotal resection, nitrogen mustard, roentgen	Alive 13 yr

SUMMARY AND CONCLUSIONS

Seven cases of pulmonary and 5 of pleural lymphomas are reported with special emphasis directed toward the pathologic aspects and comparison of these and the clinical features with previously reported material

Pulmonary resection with complete removal of the disease was accomplished in 5 patients with survival in 2 of these being over five and one-half years and in 1, one and one-third years One patient was lost to follow-up after two years and 1 died after three years from cardiac failure.

Partial resection established the diagnosis in 2 others allowing for adjunctive therapy and survival rates of eight and one-half years in 1 and thirteen years in the other

The majority of the lesions were of the lymphocytic-type lymphosarcoma and this group of patients was more favorable for resection as well as having the best survival rates

The prognosis of the lymphoma group as a whole would seem to be more favorable than that for pulmonary or pleural carcinoma

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DISCUSSION

DR. HERBERT C. MAIER, New York, N. Y.—I should like to give a follow-up on a case of small-cell lymphosarcoma that was referred to, in which the patient was operated upon ten and one-half years ago when a pneumonectomy was performed. The patient at that time was 50 years of age and (slide) had extensive involvement of the left lung including the whole upper lobe and a part of the lower lobe by infiltration across an incomplete fissure. The lymph nodes were negative. The subsequent course was as follows:

(slide) An x-ray taken three years later showed a lesion in the other lung that responded readily to small doses of radiation. He remained well until 1951, having had the pneumonectomy in 1944. A small lesion then appeared in the paramediastinal region which, as the next slide shows, increased in size.

(slide) Again with small doses of radiation, the lesion disappeared within a short time.

(slide) This is a recent inspiration and expiration film; you see there is excellent ventilation of the remaining lung which was not interfered with by the relatively small doses of irradiation. The patient has no dyspnea at the age of 61 which I think is largely due to the excellent expansibility of the remaining lung. He has been entirely asymptomatic and, as a matter of fact, did not even have symptoms at the time he showed roentgen evidence of recurrence of the disease.

(slide) I show this next case for a different purpose. This patient was operated on only three years ago for reticulum-cell lymphosarcoma. When this woman, who was in her 40's, came to us, she had x-rays that showed that the mass had almost doubled in size in a three-week period prior to operation. At operation, there was no lymph node involvement but just a large mass in the right middle lobe which involved the visceral pleura and had a granular appearance. The middle lobe was removed and she was well on a follow-up visit last week, three years after operation, in spite of the rapid growth of the neoplasm preoperatively.

DR. BRIAN BLADES, Washington, D. C.—Four cases of primary lymphosarcoma of the lung have been seen at the George Washington University Hospital. In these four cases there was one unique problem, which is probably worth describing.

The patient, a 40-year-old woman, had been using oily nose drops for many years. Her case had been used for demonstrations of lipoid granuloma for some time. She had bilateral x-ray changes compatible with this disease and fat droplets had been demonstrated in the sputum.

The internist in charge of her case had x-rays made at six-month intervals and, after a couple of years, noticed that on the right side the shadow was more opaque and was assuming a rounded, solid appearance. All her studies were repeated, but there was no clue as to what might be going on. Finally, it was decided to perform an exploratory thoracotomy. At that time a lymphosarcoma was found. No involved lymph nodes could be located in the mediastinum. Under ordinary circumstances, a pneumonectomy would have been performed, but there was a serious problem of contralateral disease, which also might be lymphosarcoma. We were reluctant, therefore, to remove the lung. Dr. Charles Thompson, the internist in charge of the case, made an excellent suggestion that we block off the side known to be involved with lymphosarcoma, with lead sheeting, and give x-ray therapy to the unknown or left side. After a test dose of x-ray there was no change in the left side, and we therefore proceeded with the pneumonectomy on the right. She is now alive and well after more than two years.

DR. ARTHUR H. AUFSES, New York, N. Y.—I think it is important to remember that in this disease there are some benign lesions. Had the essayist mentioned the first

4 cases in the literature I think he would have mentioned the 1 about which I am going to speak, which was published in the *Journal of the Mount Sinai Hospital* in 1951. This patient also had no pulmonary symptoms.

(slide) In 1947, she was found to have a lesion in the lung when she had gastrointestinal x-rays made because of some abdominal distress. The series was negative and the lesion was investigated. Two bronchoscopies were negative, and she was operated upon. A diffuse lesion was found in the upper lobe, infiltrating the middle and lower lobes. A pneumonectomy was performed because the pathologist thought this was a tumor on frozen section, although he could not say exactly what kind of neoplasm.

(slide) Here is a tomogram showing this mass; this was not atelectasis, this was tumor extending into the upper lobe. Our pathologist felt that this was what he would have to call a benign lymphoma; it was composed of a mass of mature lymphocytes which had replaced the pulmonary parenchyma and which had not invaded the bronchi or the arterial system. There were no mitoses. He could not call this lymphosarcoma as such. We decided not to give any further therapy to this patient. She is alive and well eight years after operation.

DR. JOHN P. HIGGINSON, Portland, Ore.—It is of interest to me that the authors found that bronchoscopy was of little diagnostic aid in these cases. My own experience has been very limited. In the *Journal of Thoracic Surgery* for December, 1950, Dr. Grismer and I reported an instance of obstructing intrabronchial Hodgkin's disease diagnosed by bronchoscopic biopsy. By way of follow-up report it might be interesting for the audience to know that this patient died May 31, 1953, of the disease. There was no autopsy. This was two months short of four years following diagnosis and x-ray therapy. He had been last seen with a negative bronchoscopy on July 18, 1950, twelve months following the original diagnostic bronchoscopy.

DR. JOHN L. POOL, New York, N. Y.—There are two points to which I should like to call attention. The first concerns diagnosis. We are all sure in our minds in this group, certainly, that any intrathoracic shadow, the nature of which is not clear, deserves diagnosis by histologic methods.

(slide) I would like to point out that in these shadows in the chest one should also consider the fact that a supraclavicular lymph node, even though not palpable, may furnish the diagnosis. I raise that question because of a second point, which is the question of primary treatment for lymphoma disease of the lung when diagnosis has been established. If there is no diagnosis possible by extrathoracic biopsy and intrathoracic biopsy is undertaken, it is my feeling that a minimal dissection of lung tissue is in order, because from the therapeutic point of view, radiation therapy is the treatment of primary choice. For instance, in a woman of 31 years of age, chest x-ray showed a 6 cm. spherical shadow in the anterior segment of the right upper lobe. A supraclavicular node was biopsied and showed Hodgkin's disease. Radiation therapy was employed to the neck and the localized pulmonary shadow and this slide shows the situation three years later with minimal scarring in that area, because radiation was kept to the local area. But, as also illustrated by other cases today, a new area appeared in the other lung which also disappeared under radiation therapy as the final slide shows. You see minimal scarring in both lungs and both areas are clear two years later. The conclusion is that intrathoracic surgery for the lymphomatous diseases is for diagnosis and that therapy by radiation methods is best.

DR. GEORGE W. B. STARKEY, Boston, Mass.—I would like to commend the authors for their interesting paper and briefly add 7 cases of intrapulmonary lymphomas which we (Starkey, Strieder, Lynch) gathered together recently. I agree with the authors that often supraclavicular node, pleural fluid, and bronchoscopic biopsy may fail to make the proper diagnosis and so thoracotomy should be done. This was necessary in our 7 cases.

Briefly, we had 4 cases of small cell lymphosarcoma. Two of these patients merely had biopsies and died about nine and ten months, respectively, after biopsy. The other 2 had resections, 1 being alive and well four years postoperatively [illegible] ive and well seven years later. The fifth had a reticulum-cell sarcoma, had a pulmonary biopsy, and was treated with nitrogen mustard, and is alive and well six and one-half years later. The sixth and seventh patients had Hodgkin's disease. One had a right upper lobectomy plus nitrogen mustard and x-ray and continues to do well after two years. The last patient merely had a lung biopsy and was treated with nitrogen mustard and x-ray and is also well more than two years later.

DR. JOHN M. SNYDER, Bethlehem, Pa.—I would like to add one more case which presented several interesting points. The patient, a 35-year-old woman, the mother of three children, presented herself in March, 1952, because of hemoptysis and upper retrosternal pain of one and one-half months' duration. Physical examination was negative, and x-ray of the chest showed a left upper hilar mass which in lateral view appeared slightly anterior and superior to the hilum. Bronchoscopy was grossly negative, but washings of the upper lobe bronchus were reported as showing small collections of cells resembling lymphocytes, variable in size and hyperplastic in appearance, suggesting the mass to be a lymphoma in the lung.

At exploration, the mass was found to be primarily in the upper lobe, and later pathologic examination showed it to arise in one of the primary apical divisions of the left upper lobe bronchus leading directly into the area. At the operating table what was considered to be a direct extension into the mediastinum involved the apex of the pericardium at the anterior base of the aorta, and after resection of this part of the pericardium, the mass was found also to involve the left lateral wall of the superior vena cava. The lateral wall was resected for about 1½ inches, and since a frozen section at the time from a single contiguous mass was misinterpreted as a carcinoma, a left pneumonectomy was performed. No superior vena caval compression syndrome resulted as quite an adequate amount of vein had been left. Fixed section examination of the lung mass, approximately 5 by 4 by 3 cm. in size, showed it to be a Hodgkin's granuloma. The mediastinal extension on cut section showed several matted nodes. The edges of the pericardium and superior vena cava removed with the mass were microscopically tumor free.

The patient received 2,500 r of x-ray therapy postoperatively, and as the x-ray shows is alive and well without evidence of recurrence or metastasis three years later.

DR. ROBERT J. JENSIK (Closing).—I would like to thank the discussers for their remarks, and for the addition of the cases they have seen. I am interested in what Dr. Maier said about the result in his patient. A lot can be learned by comparing end results with the histologic types of lymphomas, although there is no absolute correlation seen. In one of our cases the picture was that of a very immature lymphocytic lymphoma, and the survival was more than five and one-half years which is an exception. Our first case was Hodgkin's sarcoma and the survival of more than eight and one-half years was not in keeping with the prognosis in Hodgkin's sarcoma. So that all we can do is offer generalities which will bear support as additional material is collected.

With regard to Dr. Aufses' reference, we did not quote that, but I would like to say that making the diagnosis of benign lymphoma should be done with the fingers crossed. Several of the patients in our series had a histologic picture of very mature lymphocytes in keeping with the picture of mature lymphoid tissue. One patient of this group had disseminated lesions to the lymph nodes of the neck, inguinal region, and opposite lung at the time of death.

With regard to Dr. Pool's remarks, we always examine for peripheral nodes, because the demonstration of tumor in these in many cases precludes resection of the chest lesion. In none of the patients of this group in whom complete removal of the tumor was accomplished was peripheral lymph node involvement found. Furthermore, the hilar lymph nodes were free of any changes suggestive of lymphoma.

PROSTHESIS FOLLOWING EXCISION OF CHEST WALL TUMORS

Bert H. Cotton, M.D., George A. Paulsen, M.D. (by invitation), and
Jack Dykes, M.D. (by invitation)
Los Angeles, Calif.

SURGICAL management of 20 patients with chest wall tumors is reviewed. Criteria for chest wall prosthesis are proposed and critical areas in which replacement problems may be encountered are discussed. Stainless steel ribs or struts with fine malleable steel mesh proved to be the prosthesis of choice. Present results with this method are encouraging.

Ideal surgical treatment of chest wall tumor must accomplish two objectives: adequate excision, and return of the physiology as nearly as possible to normal. For 60 years surgeons have successfully removed large segments of the chest wall. For an equal period of time, they have been less successful in restoring normal chest wall function. The obstacles encountered are, first, the wide deforming defect: and second, the seriously altered pulmonary physiology.

A wide variety of prostheses, or none at all, will prove adequate for small defects of the chest wall. Hence the critical test of any reconstructive method is its adaptability to large defects. If successful prosthesis is to be achieved in such instances, the following criteria must be fulfilled: The unsightly deformity must be eliminated. A chest wall must be provided which is strong enough to resist blows that might injure underlying structures. This chest wall must be sufficiently rigid to prevent paradoxical motion and impaired cough mechanism and to support overlying heavy bone and muscle structures, particularly the weight of the scapula; yet it must be flexible enough to allow physiologic chest wall movements to institute proper alveolar gaseous exchange. Pain should not exceed usual expectations. Minimal bacterial contamination at operation is, of course, a requirement.

EVOLUTION OF CHEST WALL PROSTHESIS

Radical resection of chest wall tumors was recorded early in the literature. Even before 1894,[1] wide block resection of the chest wall with segments of the involved lung had been successfully performed. Although some authors[2-6] considered that repair of the defects was not essential, most surgeons have attempted reconstruction since 1894 when Volger[7] suggested the use of osteoperiosteal flaps.[8-10, 22] The first materials employed for this purpose consisted of adjacent tissue—periosteum, muscle,[11, 12] thoracic and abdominal soft tissue flaps, including the female breast,[13-15] with skin grafting if necessary. Successful results with fascia lata grafts in large sheets or woven strips have been re-

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Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 24 to 26, 1955.

TABLE I

NAME	AGE	SEX	OPERATION	PATHOLOGY	DATE OF OPERATION	TYPE OF PROSTHESIS*	ALIVE OR DEAD† (YEARS)	PREVIOUS SURGERY
J. F.	32	M	Sternum, gladiolus, and xyphoid	Reticulum-cell sarcoma of sternum	1-12-48	Tantalum plate	Dead (2)	0
M. M.	60	F	6 through 8	Adenocarcinoma of lung	6- 6-49	Mesh No. 46	Dead (1)	0
S. B.	34	F	2 through 4	Recurrent adenocarcinoma of breast	8-22-49	Mesh No. 46	Working	Mastectomy and recurrence with wide ulceration
W. M.	54	M	Lat. ½ of 2 through 6	Sarcoma mesothelioma	12- 1-50	Mesh No. 46	Dead (1)	0
F. D.	70	M	Ant. ½ of 7 and 8 and rt. pneumonectomy	Squamous-cell carcinoma	2-19-51	Mesh No. 46	Dead (1)	0
M. S.	75	M	Ant. ½ of 2 through 4	Fibromyxoma	3- 5-51	Mesh No. 46	Working	0
L. D.	48	F	Ant. ½ of 3 through 6	Adenocarcinoma of breast	11- 2-51	Mesh No. 46	Working	Bilateral mastectomy and recurrence with ulceration
O. S.	49	M	Lat. ½ of 5 and 6 plus pneumonectomy	Squamous-cell carcinoma	3-20-52	Mesh No. 46	Dead (1)	0
M. S.	16	F	Post. seg. 8 and 9	Giant-cell tumor	9- 4-52	Mesh No. 40	Working	0
M. M.	60	F	Sternum and rt. ant. 2 through 4	Adenocarcinoma	10- 6-52	Mesh No. 20	Working	Mastectomy and recurrence with ulceration
D. B.	46	F	Ant. to post. 2 through 7	Reticulum-cell sarcoma	11-17-52	Mesh No. 20	Dead (2)	0
L. M.	62	M	Ant. to post. 1 through 3 plus RUL	Adenocarcinoma	2- 6-53	Mesh No. 46	Dead (1)	0
B. V.	46	F	Diaphragm and lat. ½ of 9 and 10	Mesothelioma	4-13-53	Mesh No. 46 for diaphragm and chest wall	Dead (2)	0

J R	48	M	6 through 11, LLL, Diaphragm and spleen	Osteochondroma	12 2 53	Mesh No 20 with steel rib and polyethylene diaphragm	Working	3 attempts
J C	68	M	Ant ⅔ of 6 and 7	Fibrous dysplasia	2 22 54	Mesh No 40 and steel rib	Working	0
C B	33	M	Lat ½ of 5 through 9	Round cell sarcoma	4 12 54	Mesh No 40 and steel rib		1
			Thoracotomy	Extensive recurrence	9 27 54	Mesh No 20 and steel rib	Dead (1)	
O S	19	M	2 through 5 and RUL	Reticulum cell sarcoma	6 4 54	Mesh No 40 and steel ribs	Working	0
R H	68	M	Ant and lat ⅔ of 6 through 10 and rt pneumonectomy	Fibrosarcoma				
L B	60	F		Neurofibroma Sarcoma	12 1 39 10 11 48			Excision of neurofibroma Excision of local sarcoma with segmental lobectomy
			Post and lat ¾ of 5 through 11 and lobectomy	Fibrosarcoma	9 9 54	Mesh No 40 and steel ribs	Working	
D W	58	F	Ant ½ of 4 through 10 Post ½ of 4 through 10	Myxosarcoma	2 4 52 1 13 55	Mesh No 20 Mesh No 40 and steel rib	Working	2 attempts

*United Foundation wire mesh

†Survival rate over 6 months listed as 1 year

ported.[16-18] Later, repair of chest wall defects was accomplished by means of adjacent ribs either split or divided in such a way that they could be pulled down over the defect. Tibial grafts,[19] or rib and cartilage in strut or diced form were employed also.

In 1909,[20] metallic prosthesis was proposed. Tantalum plates[13, 17, 21-27] were the first metallic replacements used in the chest but were largely unsuccessful[17, 22-27] because of complicating hemorrhages and infections. Regardless of the substance used and even in the absence of tissue reaction, it appears unlikely that a fixed plate could become an integral part of the supporting tissues of the chest wall. A smooth plate resists intimacy with surrounding tissue. When the involved structures are subject to constant changes in stress the possibility of union is still more remote.[22, 23, 28] Tantalum mesh has been used with success to repair chest wall defects as well as in other parts of the body.[8, 28-31] In a recently reported series of cases,[28] however, a change from this material to stainless steel wire was made after late fragmentation of the tantalum mesh occurred. Owing to excessive tension, 2 patients sustained local skin necrosis which produced separation of the wound down to the mesh. The necrotic area healed by granulation without sinus formation.

AUTHORS' SERIES

Statistical data concerning the 20 cases comprising the present series are summarized in Table I. Three patients with benign tumors are alive and well an average of 3 years and 6 months after operation. Of 10 patients with primary malignant tumors of the chest wall, 5 are alive an average of 3 years and 3 months after operation while 5 died an average of 1 year and 7 months postoperatively. Metastatic extensions of breast carcinoma were present in 3 patients, all of whom are well an average of 4 years and 4 months after surgery. Four patients with metastatic extensions of lung tumors died an average of 13 months postoperatively. There were no deaths 6 months after operation in this series.

TABLE II. CHEST WALL TUMORS

	ALIVE		DEAD	
	NO. CASES	AVERAGE YEARS OF SURVIVAL	NO. CASES	AVERAGE YEARS OF SURVIVAL
Benign	3	3.6	0	
Malignant	5	3.3	5	1.7
Metastatic	3	4.4	4	1.1

TABLE III. TYPE PROSTHESIS

Tantalum plate	1 case
Steel mesh	13 cases
Steel mesh with steel rib	6 cases

Insofar as possible, excision and prosthesis for chest wall tumor should be considered separately; otherwise, a compromise resection of the tumor may result.

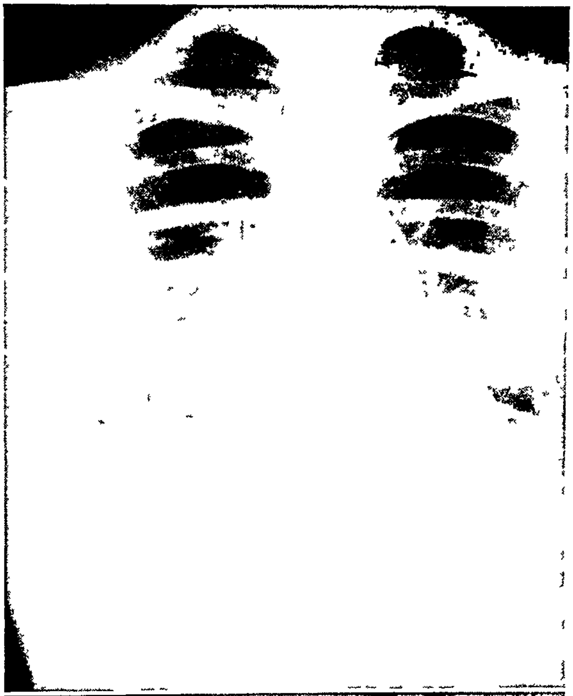
Resection.—Adequate resection requires excision of the tumor and involved rib or ribs together with a safe margin of adjacent tissue. In our series this margin included 3-inch segments[32, 33] of ribs beyond the tumor. Intramedullary extension may occur as far as 3 inches into the involved bone. Furthermore, microscopic nodules of the tumor have been found in distant periosteum and recurrence in the rib stumps has not been uncommon. The margin should include intercostal muscles above and below, a procedure executed with minimal danger of transecting tumor nodules by removing segments of adjacent ribs, underlying parietal pleura with lung tissue if involved, and immediate overlying skeletal muscle with skin, if involved, en bloc. This type of resection is a minimum for malignant tumors.[17, 26, 35] Other structures may be included as indicated.

Prosthesis.—An arbitrary indication for prosthesis is the removal of two or more long rib segments. Re-establishment of chest continuity should then be attempted in accordance with the previously mentioned criteria. Three areas exist in which it is most difficult to accomplish effective stabilization. The first critical area is the anterior chest wall where lack of overlying soft tissue and wide interspaces present a problem. Second, resections extensive enough to undermine the supports of the scapula necessitate a replacement that will support the heavy structures of the back. Third, if the excision extends through the lower intercostal margins, a substitute must be envisioned to maintain support for the lower border of the chest wall, pull of the diaphragm and the liver or stomach.

Our experience began in 1948 with an operation for a reticulum-cell carcinoma of the sternum. A longitudinal incision was made over the lower border of the chest and a skin flap dissected upward so that the line of incision would not be over the prosthesis. The surgical excision required removal of a portion of the manubrium, the entire gladiolus, ziphoid process, and segments of the cartilaginous ribs on both sides of the sternum. A large tantalum plate was fitted to the defect and sutured in place with tantalum sutures. The plate previously had been drilled with many perforating holes so that it could become firmly anchored by tissue growth. Entrance into the right pleural space was made during the procedure. The pneumothorax was effectively controlled postoperatively by an intrapleural catheter. Postoperative results in this instance were satisfactory. The plate became firmly fixed and the wound healed per primam. Excellent protection to the anterior mediastinum was achieved. Success was credited to the many perforations in the tantalum plate which rapidly became solidly anchored in the tissue.

This type of replacement, although it may still be considered applicable for the sternum, presents certain disadvantages. The tantalum plate proved difficult to mold to the sternal defect and would be much more difficult to mold to the curvature of the ribs. Moreover, the material is unnecessarily heavy. Because of favorable reports in the literature and our own experience with stainless steel suture material over tantalum, we decided to try stainless steel mesh in subsequent cases. On June 6, 1949, the stainless steel mesh was first

Fig 1.



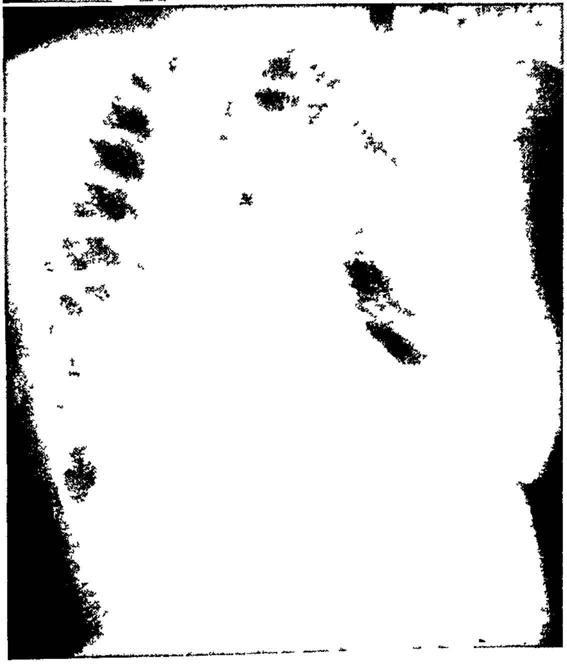


Fig 2.

Fig. 1—J F Tantalum plate replacing sternum Close scrutiny shows holes bored hrough plate which aided fixation of the prosthesis

Fig. 2—D B Preoperative view of sarcoma on right posterolateral chest which has roded right fifth rib and portions of the fourth and sixth ribs

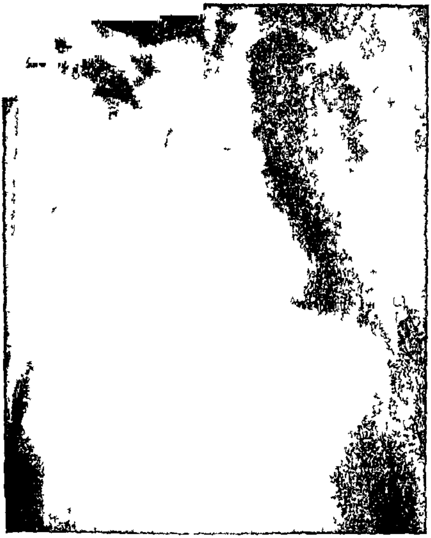


Fig 3 —D P Same patient postoperatively displaying buckled plate of unsupported mesh

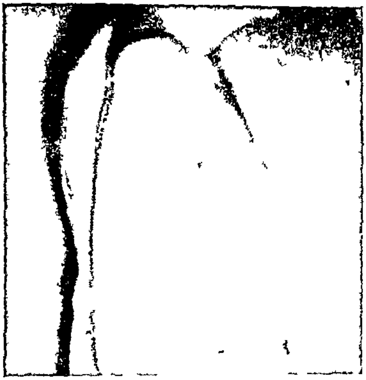


Fig 4

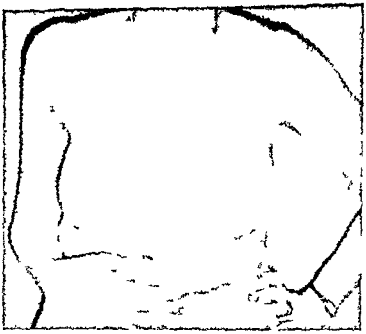


Fig 5

Fig 4 —D B Lateral view showing sunken right chest wall due to scapula weight on steel mesh inadequate for supporting shoulder weight

Fig 5 —D B Anterior view showing fixed right scapula Shoulder weight too great for unsupported mesh Anteriorly with no weight to buckled prosthesis shows no deformity

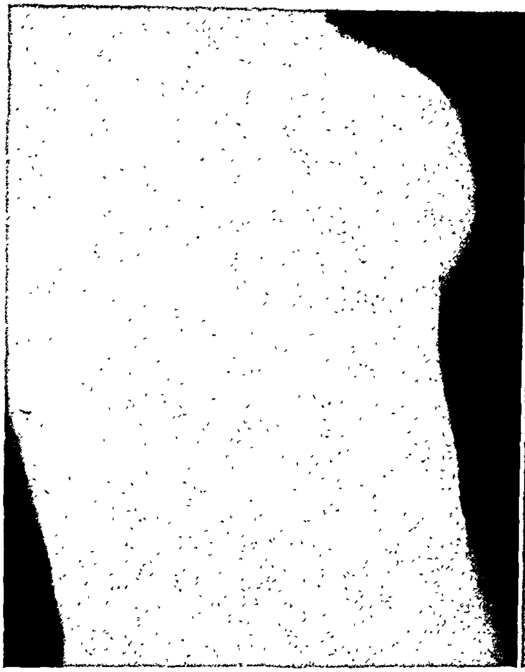


Fig. 6.

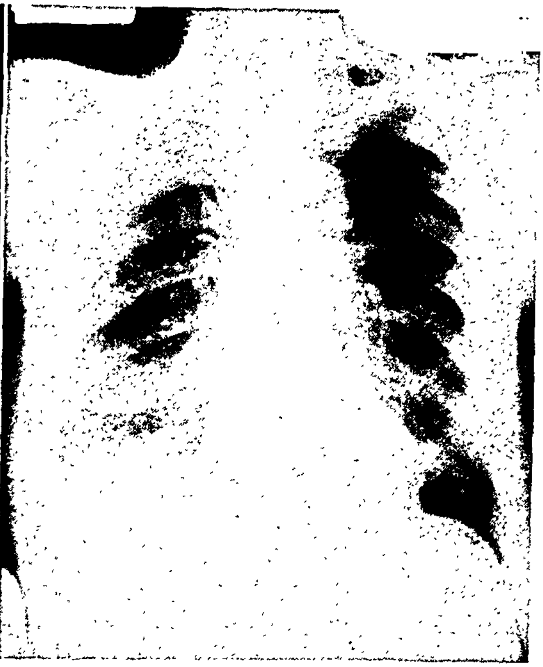
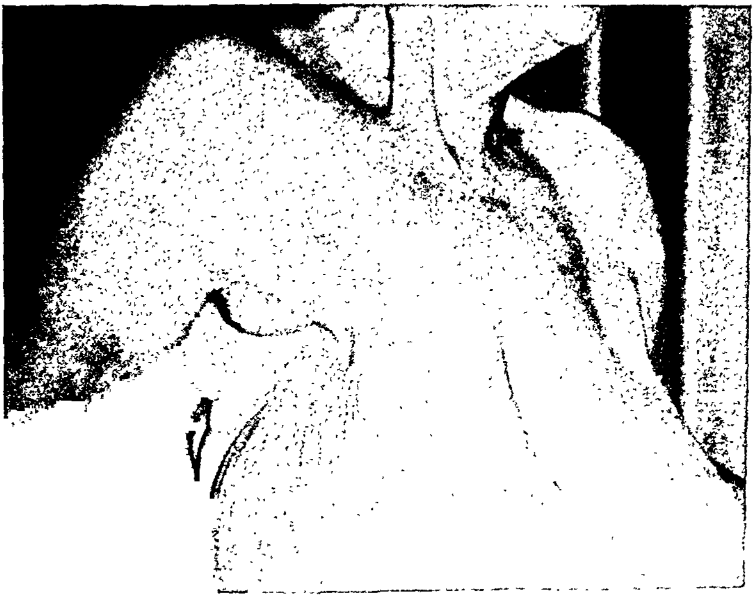


Fig. 7.

Fig. 6.—M. S. *Preoperative film (of soft tissue technique) showing tumor on anterior chest.*

Fig. 7.—M. S. Same patient with prosthesis of stainless steel mesh replacing portion of right anterior chest wall.



ig. 8.—D. W. Example of deformity from depressed chest wall. This patient had an infection of space about prosthesis requiring removal of the heavy mesh.

employed following a right pneumonectomy for adenocarcinoma A block resection of the posterior chest wall was necessary to remove the lung Segments of the sixth, seventh, and eighth ribs were excised A single sheet of No 46 mesh was used successfully This material was later employed on several occasions and appeared to provide adequate reinforcement of the chest wall

On March 5, 1951, we first experienced difficulty with stainless steel mesh following a large block resection of the anterior chest wall which included the second, third, and fourth ribs The support proved too weak to form a rigid replacement for such a large defect Limited paradoxical motion occurred with depression of the replacement toward the lung, causing the edges of the overlapping mesh to exert some pressure on the skin The result was adequate but not ideal Even today the area demonstrates mobility when the patient coughs

In order to lend added strength to the prosthesis, we adopted a heavy-weight stainless steel screen wire No 20 in subsequent cases This regulation screen wire proved very satisfactory for several large anterior defects and probably would have served as well as the tantalum plate in resections of the sternum In the remaining two critical areas however, it was unsatisfactory Following an excision block including the inferior costal margin the screen wire was molded to the curve of the ribs where it was sutured because of its springlike tendency The sutures broke whereupon the unanchored edge sprang out causing so much pressure pain and skin deformity that its removal was necessary The heavy weight screen wire has no resilience It further proved unsatisfactory for the critical subscapular area where a large sheet was used following an extensive chest wall dissection of the entire second third, fourth, fifth, and sixth ribs with the lateral half of the seventh which undermined the support of the scapula and heavy back muscles Despite the heaviness of the mesh No 20 and the outward arch which added to its strength, the posterior portion buckled and allowed the scapula to be displaced inward causing a severe posterior deformity Another disadvantage of the heavy mesh support is its size and weight, in this respect it is similar to tantalum plates, a slow resisting intimacy of the surrounding tissue Thus immediate postoperative infection will always require removal of the prosthesis No such problem was encountered in reported cases in which fine mesh was employed [7,8]

Our next attempt to design a satisfactory prosthesis concerned a recurrent chondroma weighing 22 pounds which occupied the entire lower half of the left chest and necessitated removal of the sixth, seventh, eighth, ninth tenth and eleventh ribs, left lower lobe spleen, left diaphragm, and the greater portion of the lumbar muscles A special prosthesis was made from heavy stainless steel mesh with supporting stainless steel ribs Unfortunately, this did not fit the defect perfectly and was extremely awkward to refashion at the operating table Because of extensive skin flaps heavy weight of prosthesis, and tension of the suture line, necrosis occurred, uncovering the mesh Widespread infection required removal of the prosthesis

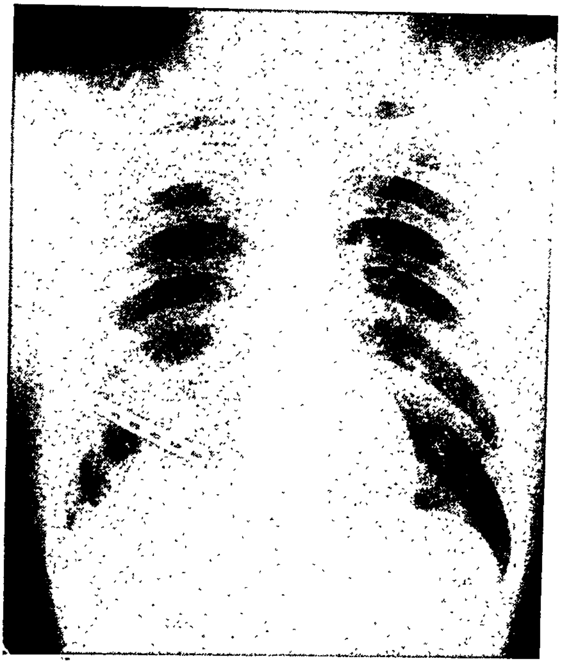


Fig. 9.—C. B. One strut support for mesh prosthesis.

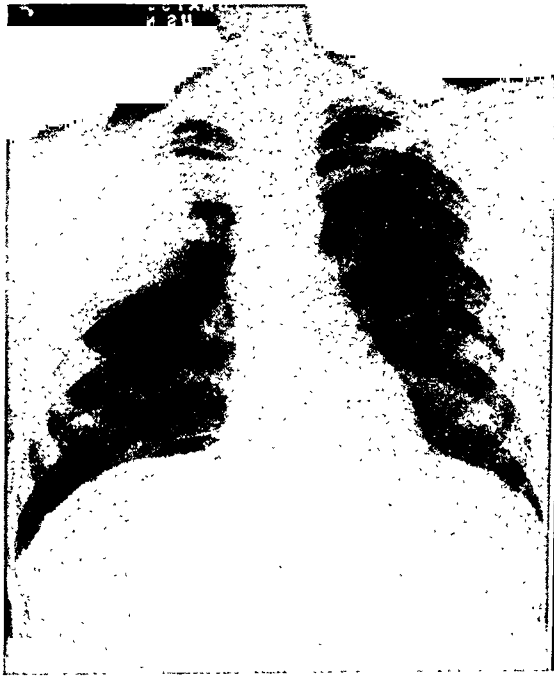


Fig. 10.

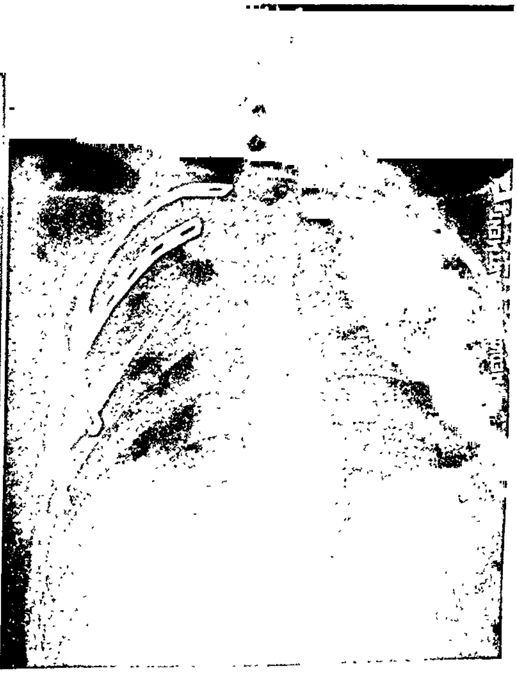


Fig. 11.

Fig. 10.—O. S. Preoperative view of right chest wall tumor which has dissolved lateral portion of right third rib.

Fig. 11.—O. S. Prosthesis of two-rib strut support to fine mesh which was fashioned at operating table to tailored size.

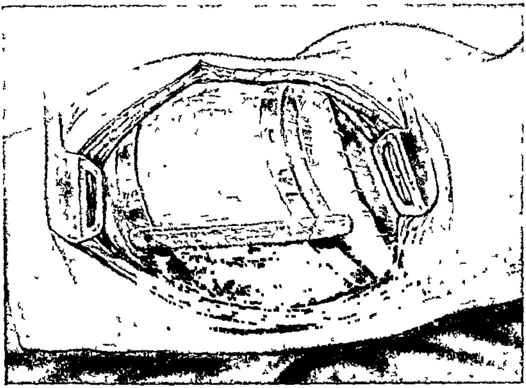


Fig. 12—Strut prosthesis in place showing malleable mesh supported by stainless steel rib supports

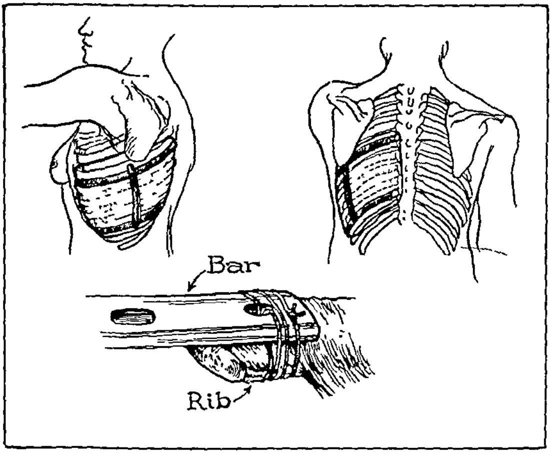


Fig 13—This view shows method of attachment of strut to adjacent normal rib end It also shows lack of deformity

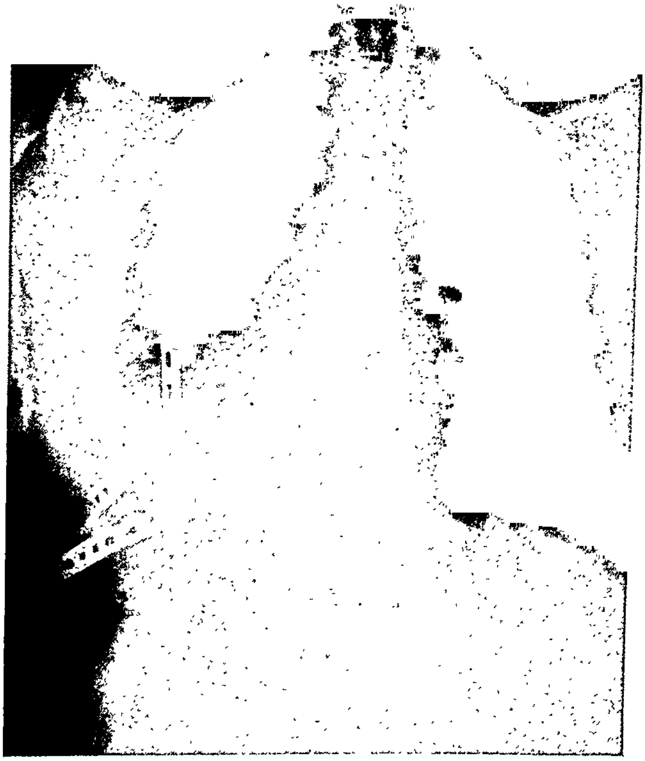


Fig. 14.—L. B. Three-rib strut support to fine malleable mesh with one-rib strut as cross strut.

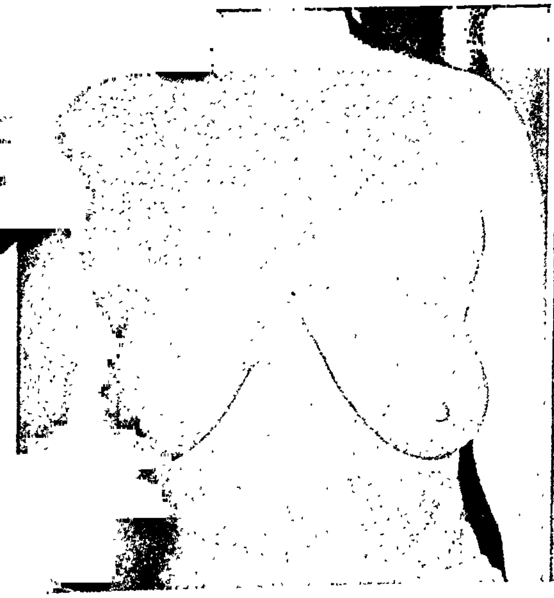


Fig. 15.

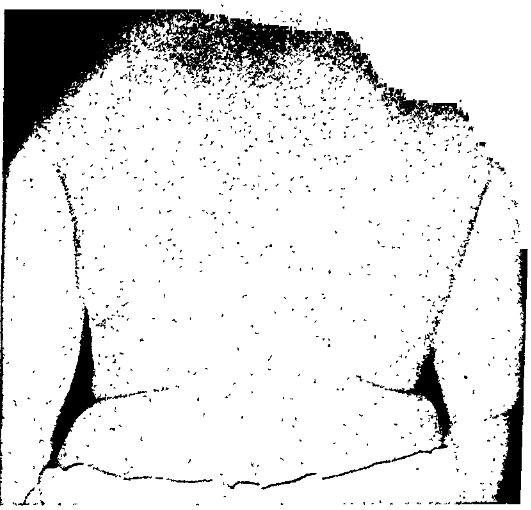


Fig. 16.

Fig. 15.—L. B. Anterior view shows symmetrical-appearing chest wall with large section replaced by prosthesis described.

Fig. 16.—L. B. Posterior view of same patient.

The present method, evolved from our experience with chest wall replacements, consists of stainless steel rib struts, about one for every two ribs removed, placed strategically over the defect and anchored to the posterior and anterior portions of the ribs A perpendicular cross strut may be used to provide additional strength This scaffold will perform the weighty duties of the thorax A fine malleable mesh placed over or under the frame of the strut acts as matrix and lattice for tissue growth and performs the less strenuous task of resisting intrapleural pressure changes Such a pattern allows the surgeon considerable leeway for construction at the time of operation owing to the various lengths of orthopedic bone plates available as struts and the malleability of the fine steel mesh which is integrated into the tissue rapidly with no pressure points We have encountered no complications with this type of prosthesis and postoperative results have been satisfactory As compared with previous techniques we feel our present method more fully meets the criteria for a successful prosthesis It more nearly duplicates the normal anatomy of the chest wall and provides a better physiologic functional chest with adequate mobility but with sufficient rigidity for support By clinical application we have established that this type of prosthesis will meet the demand for scapula posteriorly, will effectively stabilize the chest anteriorly, and will substitute for a lower costal border, that is, it serves satisfactorily in the three critical areas previously defined It may be used effectively to stabilize the lower costal edge after resections of the lower chest extending through the inferior costal border to the abdominal muscle wall If the attachment of the diaphragm must be dissected, it may be reattached through the screen to the underlying muscle or skin tissue adjacent to one of the supporting steel struts Diaphragmatic action thus is not lost The prosthesis therefore meets the critical test of adaptability to large chest wall defects, including those in which stabilization is most difficult to accomplish by other methods

COMPLICATIONS

The complication encountered in this series was infection, occurring in 2 patients both of whom had prostheses made of heavy mesh In one of these patients the skin necrosed at the incision line owing to extensive skin flaps and tension of suture line Heavy prosthesis was considered the cause Contrary to reported observation in patients in whom fine mesh was employed,[8] a widespread infection was encountered as soon as the screen became visible This may be related to the lesser facility with which the heavy screen wire is incorporated in the tissues The second instance of infection occurred after an extensive chest resection including ribs four, five, six, seven, eight, nine, and ten, accompanying a lower lobectomy A temporary bronchopleural fistula was the origin of infection In both cases it was necessary to remove the prosthesis in order to control the infection and then to make a fresh start toward eliminating the defect

SUMMARY

1 The results of excision and prosthesis in 20 cases of chest wall tumor are presented

2. Criteria for the prosthesis are suggested and critical areas which may prove difficult to manage satisfactorily are pointed out.

3. A new type of prosthesis is proposed which appears to fulfill essential criteria. The replacement, consisting of stainless steel ribs with fine malleable steel mesh, proved satisfactory in critical areas in the patients to whom it was applied.

4. The final test of a prosthesis is its adaptability to large defects. A variety of replacements or none at all will be satisfactory for smaller defects.

The authors wish to express their thanks and appreciation to Mrs. Helen Gindy, Librarian of the Huntington Memorial Hospital, Pasadena, Calif.

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DISCUSSION

DR. RALPH A. DETERLING, Jr., New York, N. Y.—I have been interested in chest wall replacement for several years because of the occasional patient with respiratory obstruction from pressure by an aortic aneurysm. We usually "floated" the manubrium by resecting the adjacent attachments, but did work on Lucite replacements in the laboratory. This could be molded to shape with heat, and it was well tolerated in dogs.

We have also used a taffeta fabric of synthetic material in patients to fill defects of the chest wall. It is easily cut to shape and, if sewn under some tension, the patch renders support. The ingrowth of fibroblasts soon stiffens the material Should contour be desired, one can use the twelfth rib of the patient or homografts from the bone bank. I think the results mentioned by the authors are commendable, and present these other methods only to show additional ways in which defects of the chest wall may be handled.

In the 19 cases of thymic tumor which are presented, 16 proved to be malignant. The diagnosis of thymic neoplasm was established in every case by excision of the tumor or adequate tissue biopsy. The factor of malignancy was determined on the basis of the gross characteristics and the histopathologic features of the neoplasm. The criteria of malignancy in this series may be stated simply: (1) direct extension through the capsule into adjacent tissue, (2 intrathoracic implantation, (3) recurrence, and (4) distant metastases.

TABLE II. MALIGNANT FEATURES IN 16 THYMIC TUMORS

CRITERIA	NO. OF CASES
1. Direct extension	10
2. Pleural implantation	4
3. Recurrence	1
4. Distant metastases	1
TOTAL	16

By way of explanation, the listing in Table II is based on the single outstanding feature in each case (actually the majority presented more than one). The patient with implantation of a pleural space and secondary effusion has by necessity direct extension of the neoplasm through the capsule and adjacent pleura. Our tabulation does not describe the curious factor of localization presented by malignant thymic tumors. Only one patient demonstrated a distant metastasis (Case 16, K. Y.); a submaxillary lymphatic metastasis appeared several months prior to death. It is also of interest that 2 patients (Case 2, G. H.), and Case 4, F. L.) were operated upon for small encapsulated tumors that were considered benign in every respect. After apparently successful surgery, both patients demonstrated extensive implantation 3 years after operation (neither had received radiation therapy after surgery) (Fig. 1).

In this series, the resected neoplasms were for the most part not large, 12 cm. being the greatest diameter encountered. Grossly, the tumors often appeared to be encapsulated by the contiguous mediastinal tissues and anterior pleura removed with them. The cut surfaces were not in any way distinctive for any cell type and a wide variety of configurations were seen. Two of the neoplasms were cystic; one (Case 2, G. H.) so completely so that only small cell nests could be seen beneath a partially calcified capsule, and at that time, a definite histopathologic classification was not possible. This patient returned 3 years later with local and intrapericardial metastases. The solid tumors had varied homogeneous, granular, or bosselated surfaces. They were relatively firm and ranged in color from white through various shades of yellow and tan to a salmon pink.

In addition to those features previously mentioned, the neoplasms possessed certain histologic features in common. Many of the tumors demonstrated a marked tendency to incorporate within their confines the surrounding fatty tissue and to show evidence of subendothelial bulging of the wall in small veins of the immediately adjacent tissue (Fig. 2). The significance of this latter is not completely understood unless it bears the same connotation as is found in such neoplasms as the encapsulated *angio-invasive* carcinoma of the thyroid.

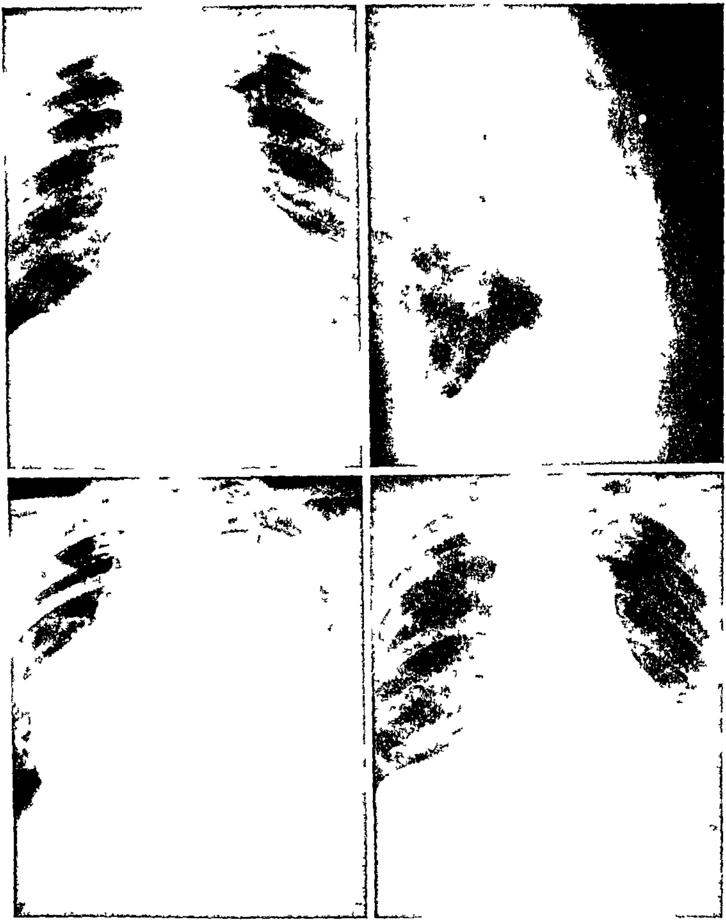
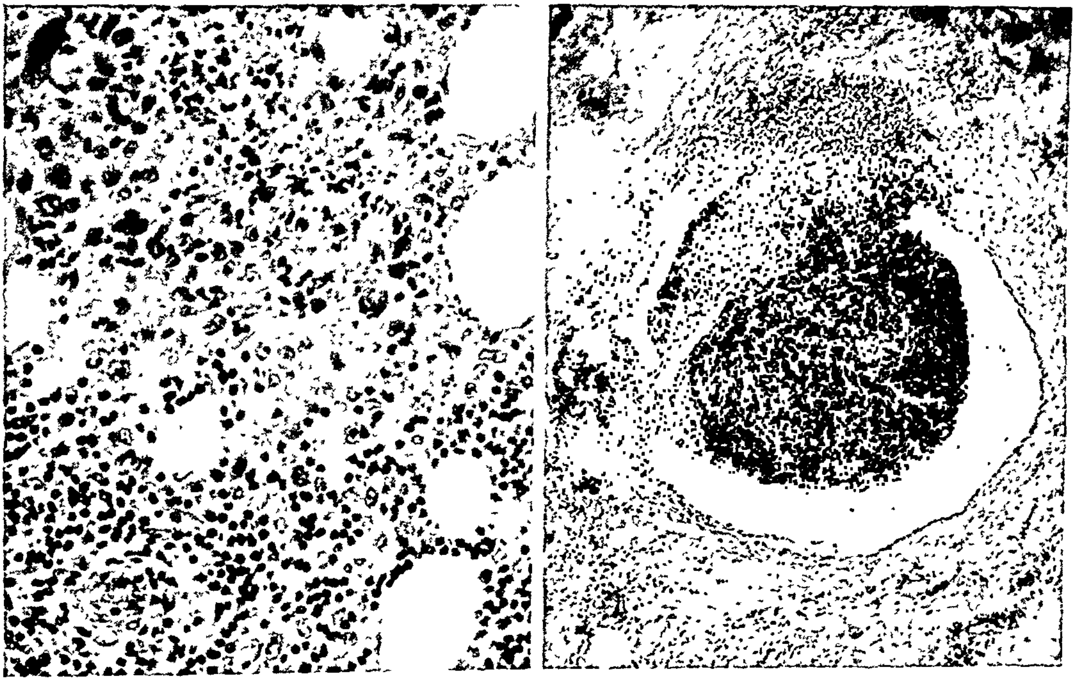


Fig 1 —(Case 2 G H) The roentgenograms at top demonstrate the thymic neoplasm of the anterior mediastinum as it appeared in July 1949

Lower left is the reproduction of film made in November 1952 The tremendous cardiac silhouette is produced by a massive effusion secondary to interpericardial implants Evacuation of the effusion and biopsy of the interpericardial implants were performed by surgical pericardotomy

Lower right is reproduction of film taken March 1955 two and one half years after radiotherapy The patient remains clinically well

It obviously must represent some mechanism of farther growth and spread of the neoplasm; however, gross intravascular extension is not a feature of these tumors. Apart from the cystic neoplasms encountered and the Hodgkins subgroup to be discussed, necrosis was not a feature of these tumors. The stromal arrangements were quite similar in most respects; broad bands of collagen tended to subdivide the neoplasms into smaller lobules. For the most part, this connective tissue was rather dense; but in 2 cases, focal areas showed what we have designated as stromal hyperplasia. Small spindle cells were greatly increased in number in the interstices of the 2 neoplasms and at times appeared to invade the adjacent tumor tissue proper and to form small whorls. Lowenhaupt[2] has considered these cells to be a form of epithelial proliferation, but we are more inclined to look upon them as abnormalities of the stroma. With phosphotungstic acid—hematoxylin stains, the individual cells possessed fine fibrils, compatible with those of fibroblasts, and in the maturing areas, deposition of collagen was a constant feature as demonstrated by the Masson's polychrome stain. This stromal hyperplasia can be very confusing. We recently encountered a case in which stromal hyperplasia was so marked that fibrosarcomatous change was suggested. The lesions in general showed a great deal of mitotic activity scattered throughout the parenchyma.



A. B.

Fig. 2.—Microscopic evidence of aggressive nature of the tumors.

A, (Case 10; O.R.) Photomicrograph showing invasion of fatty tissue by Hodgkin's variety. (Hematoxylin and eosin ×285; reduced 1/6.)

B, (Case 13; W.L.) Photomicrograph demonstrating subendothelial bulging by lymphocytes in marginal condensation of fibrous tissue (capsule) of neoplasm. (Hematoxylin and eosin ×80; reduced 1/6.)

TABLE III NEOPLASMS OF THY[illegible]

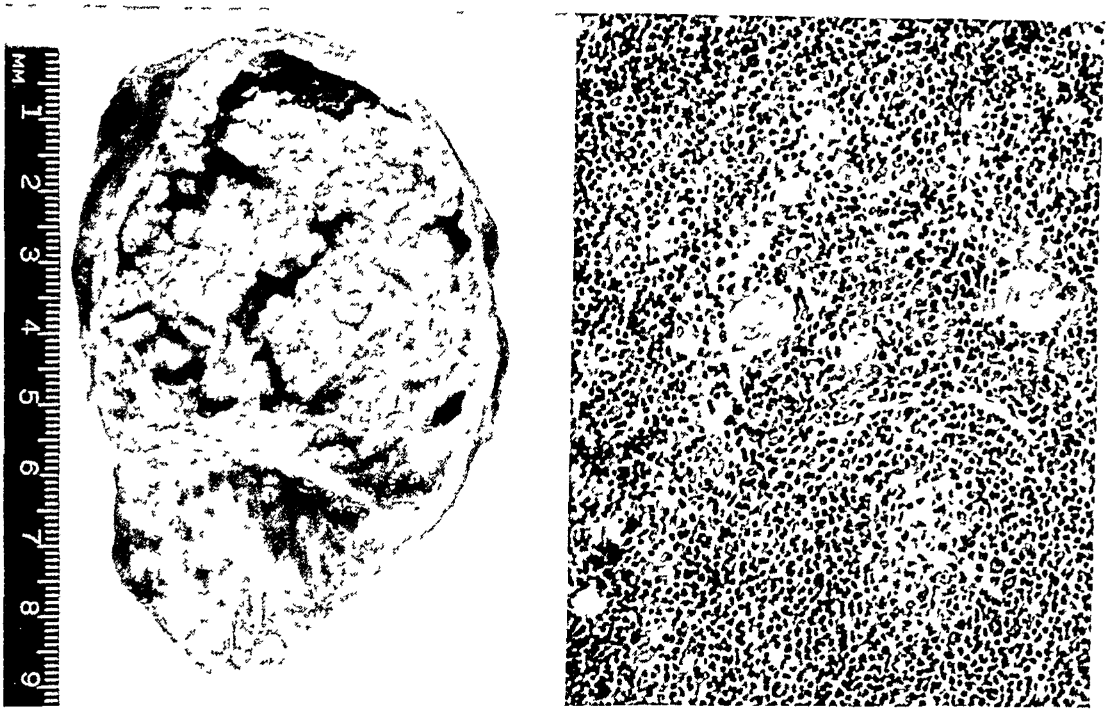
	[illegible]
1 Lymphomatous type	
a Lymphocytic variant	6
b Hodgkin's variant	[illegible]
2 Carcinomatous type	
a Lympho epithelioma	[illegible]
b Epithelial variant	1

A classification of the histopathologic features [illegible] of thymic neoplasms has intrigued pathologists for man[illegible] of Margolis[4] offers an example of the difficulties encountered [illegible] fication Lowenhaupt[5] has been the most recent of the writers to [illegible] involved table of nomenclature Seybold and coworkers,[illegible] Ackerman[illegible] and [illegible] find her classification interesting, but of relatively little value so far as the neoplasms are concerned We do believe, however, that one [illegible] be more definite in classification and not be limited to the observation that neoplasms are either predominantly lymphocytic or predominantly epithelial We agree with the general statements that neoplasms are very difficult to categorize, as they show a reluctance to present themselves as pure forms which can be easily compartmented The problem of being able to distinguish between the benign and malignant thymoma on a histopathologic basis is very real, all have the ability to be aggressive regardless of the classification groups Consequently, we have considered them all as of malignant potential and believe that malignant *thymoma* should be the term utilized for general classification

We have divided the malignant thymomas into 2 subgroups (1) the lymphomatous type, and (2) the carcinomatous type In so doing we have been content to accept the theories that the thymus is an endodermal derivative basically possessing a supporting framework of epithelial cells which may become rather reticular in appearance The lymphocytic component represents secondary infiltration of the organ and the small round cells are truly lymphocytes

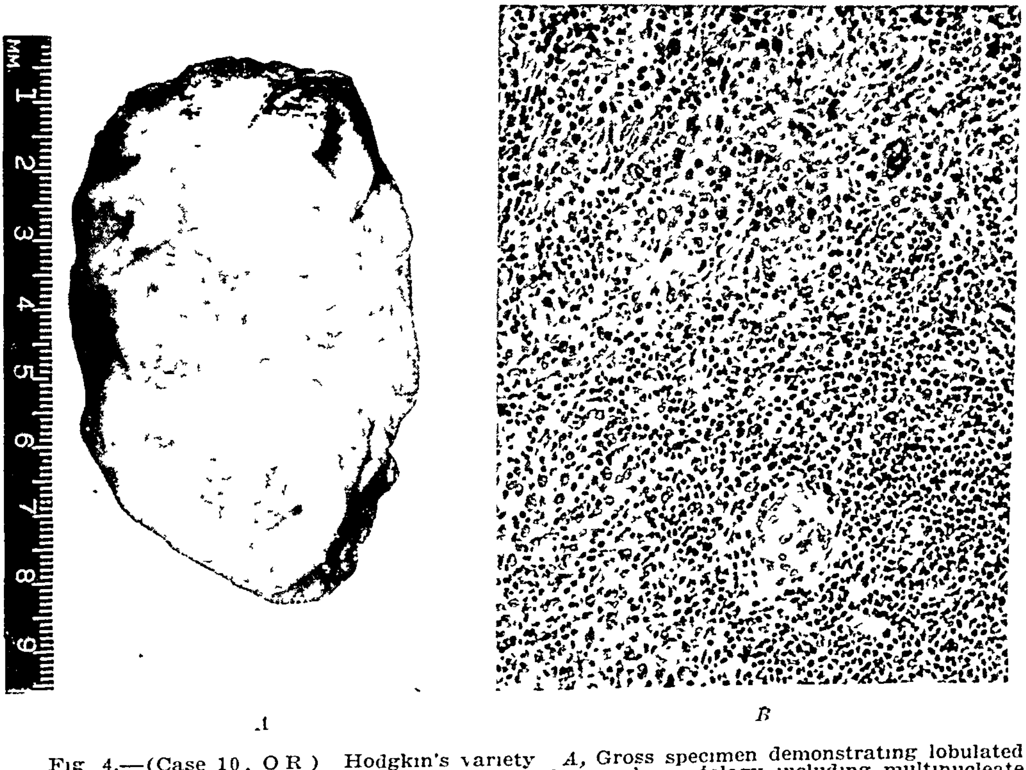
1 *Lymphomatous Type*—Nine of the cases have been classified in the lymphomatous subdivision, that was further subdivided into the lymphocytic and the Hodgkin s type In 6 of the cases, the cellular component encountered was almost completely lymphocytic (Fig 3 We do not say that the neoplasms were completely lymphocytic, as one may encounter in the background an occasional reticular cell, small proliferative cell nests resembling epithelial cells, to be described later, occur along the blood vessels We have not been disturbed by their presence as they represent a true component but are comforted by them as they constantly indicate that the thymus was the source of the neoplasm

The second subdivision of the lymphomatous group encompasses 3 cases catalogued as malignant thymoma *Hodgkin s type* (F[illegible] 4) In our earlier experience we can recall certain neoplasms fatal in their outcome, that were apparently limited to the anterior mediastinal structures which we co[illegible] as a localized form of Hodgkin s disease It is likely that these [illegible]



A. B.

Fig. 3.—(Case 7; N G) Lymphocytic variant. *A*, Gross tumor is predominantly cystic with necrotic wall The viable homogeneous portion is at inferior pole *B*, Photomicrograph showing tumor composed predominantly of small lymphocytes Rare abortive Hassall's corpuscles included to show their presence. (Hematoxylin and eosin ×210; reduced ⅙)

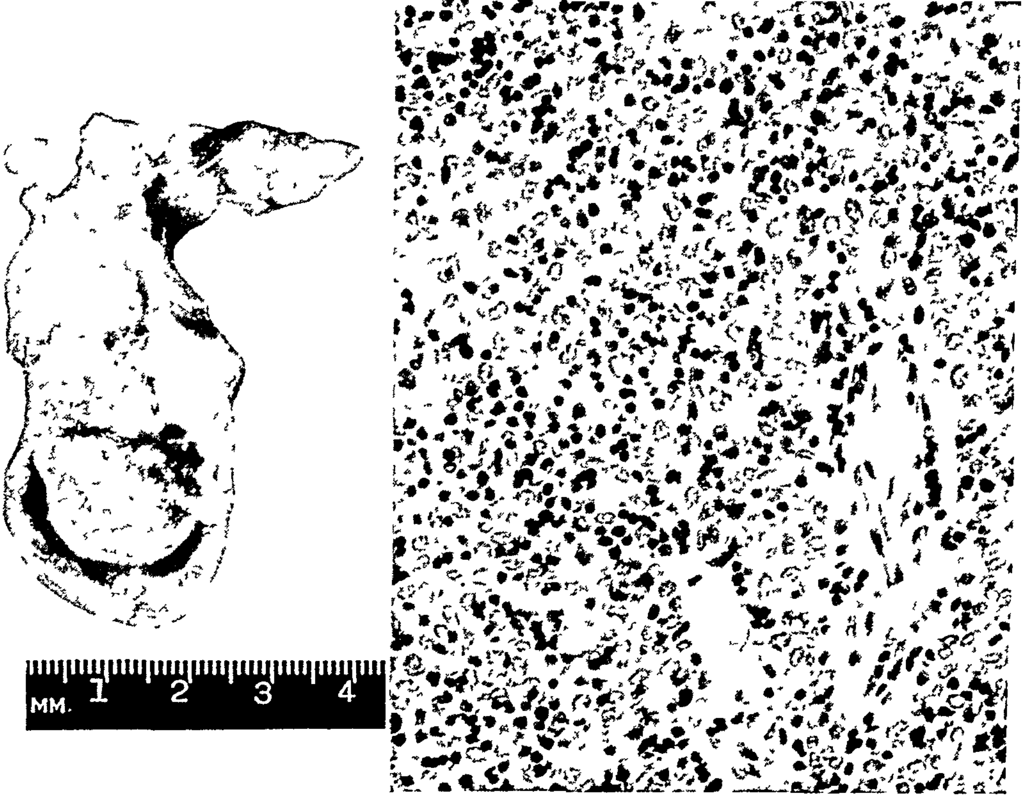


A B

Fig 4.—(Case 10, O R) Hodgkin's variety *A*, Gross specimen demonstrating lobulated cut surface *B*, Photomicrograph demonstrating pleomorphic cytology including multinucleate Sternberg-Reed cells Note bizarre Hassall's corpuscle in lower portion of field. (Hematoxylin and eosin ×160, reduced ⅙.)

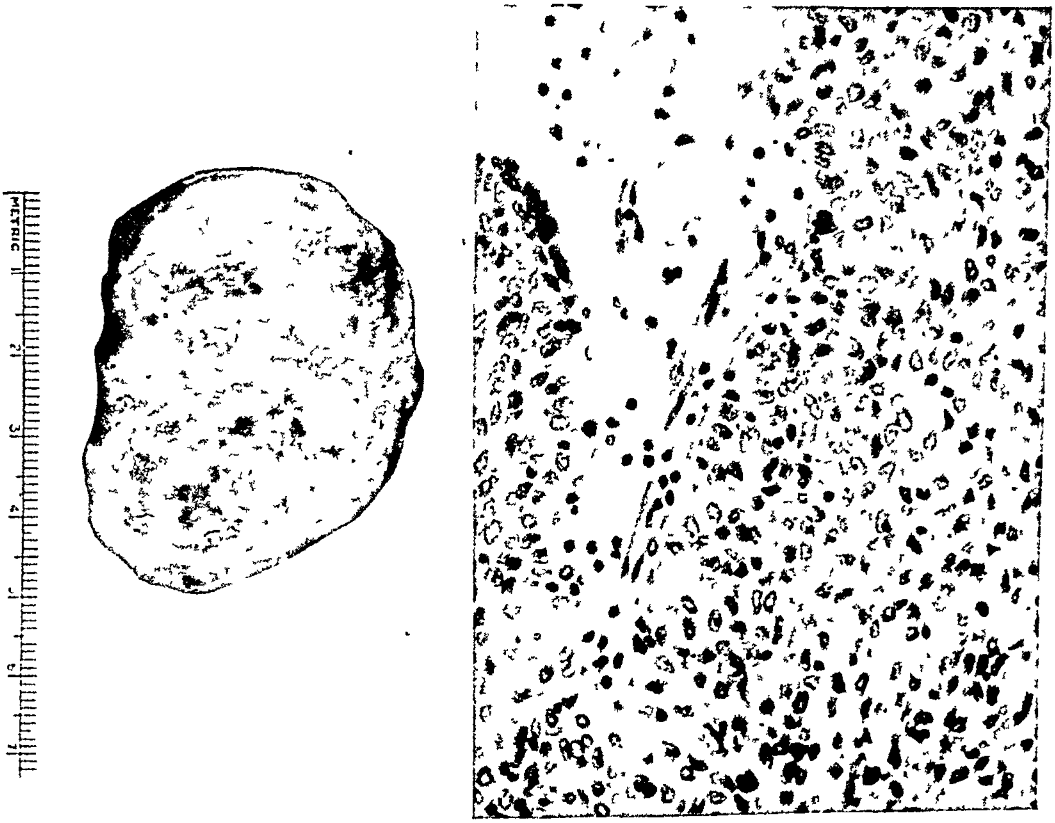
examples of the disorder limited to the thymus. All of the present 3 cases show histologic features in common. Characteristically, the tumors are made up of conglomerate foci that appear to be passing through certain histologic changes. The earliest features seemed to be the formation of cellular aggregates surrounded by small lymphocytes. These cellular aggregates superficially resembled germinal centers, but are characterized by a markedly polymorphous cytology. Cells with sparse cytoplasm and irregularly oval or lobulated nuclei, and at times prominent nucleoli, are common. In addition, multinucleated giant cells with more abundant gray cytoplasm and vesicular nuclei, indistinguishable from Sternberg-Reed cells, were found. They show an absence of any staining properties of their cytoplasm by the periodic acid Schiff method. Later, central necrosis apparently occurs within the cellular aggregates, and macrophages with more abundant pink-staining cytoplasm and round or oval vesicular nuclei appear. Still later, deposition of collagen is present in the necrotic areas. The necrotic stage superficially resembles granulomatous change and hence the term "granulomatous thymoma," a rather poor designation for the neoplastic disorder. These neoplasms, however, still possess the propensity to form bizarre Hassall's corpuscles. The source of this particular subgroup is again a philosophic question at the present time. Whether they are a peculiar transformation of the supporting cells of the thymus or truly lymphomatous neoplasm is a point of debate. We have favored the latter by designating it as a Hodgkin's variety.

2. *Carcinomatous Type.*—The second major subdivision of malignant thymomas, the carcinomatous variety, is characterized by prominence of the thymic epithelial cell. There appear to be 2 subvariants of the carcinomatous type: (1) lympho-epithelioma, and (2) the epithelial variant. The lympho-epithelial group contains 6 cases, all of which were characterized by a definite admixture of true cytologic forms (Fig. 5). The small, round, densely staining lymphocyte was ever present in varying amounts and intermingled with cells possessing round, oval, or slightly lobulated vesicular nuclei, and only a sparse amount of cytoplasm. This epithelial cell, although present in large numbers, may at times be rather small and not quickly recognized, but at other times is rather large in proportion to the lymphocyte. They are dispersed in small nests, larger sheets, and at times are quite prominent along small vessels. The epithelial carcinomatous variety is merely one step further along the scale. Four cases have been catalogued as belonging to this group. The neoplasms are characterized by the presence of large syncytial masses of cells forming the entire neoplasm (Fig. 6). A moderate amount of pink-staining cytoplasm is present, but cell boundaries are very indistinct. The nuclei are round or oval, and pale staining for the most part. The continuous masses are usually sharply demarcated from the small vessels and there is a tendency for the nuclei to palisade around the vessels. We have been unable to substantiate any evidence of cyst formation within this group of neoplasms. Lymphocytes still are present but are very sparse in number and merely scattered discriminately throughout the tumor. Occasionally these tumors do show evidence of having Hassall's corpuscle formation.



A *B*

Fig. 5.—(Case 1, G. L. H.) Lympho-epithelial variant. *A*, Gross specimen is partially necrotic and cystic, radiation therapy applied two and one-half years prior to removal. *B*, Photomicrograph of mixture of small, dark-staining lymphocytes and larger indistinct cells with pale round or oval large nuclei. (Hematoxylin and eosin ×285.)



A *B.*

Fig. 6.—(Case 11, M. C.) Epithelial variant. *A*, Small apparently encapsulated neoplasm with faintly lobulated surface. *B*, Syncytial is sharply marginated from vascular stroma. A suggestion of peripheral palisading is present; note absence of cellular outlines. (Hematoxylin

We recognize that this classification may be an arbitrary one and that all of these neoplasms may represent segregated stages of the same process However, it does lend order to our clinical and histopathologic findings As a classification, *it offers little of prognostic significance It is our hope that it* may contribute to the most important phase of the problem, recognition that a tumor is of thymic origin and with malignant potentiality

TREATMENT

The presence of myasthenia gravis is not the primary indication for therapy, *it does impose an added hazard and*, in some instances, an insurmountable barrier to tumor eradication Treatment of a thymic neoplasm as a phase in the therapy or the prevention of myasthenia gravis is of secondary import The essence *of therapy in thymic neoplasm, as* in all mediastinal tumors, should be directed toward cancer control

There are 3 basic approaches in the management of thymic neoplasms (1) biopsy, (2) *surgical excision, and* (3) radiation therapy In each case 1 or all 3 approaches to therapy are indicated

Biopsy—Diagnosis of a primary thymic neoplasm is dependent on histo*pathologic examination* The *ideal method of obtaining tissue is by* extracapsular "enucleation" of the entire mass When there is radiographic evidence of implantation, or there is evidence of malignant extension by superior caval *obstruction or penetrating* chest wall *invasion, a* less *inclusive form of biopsy* is indicated Such biopsy requires a direct approach by one of three methods (1) thoracotomy or suprasternal incision, (2) needle biopsy, or (3) transsternal biopsy We have used *a limited anterior thoracotomy approach* under local anesthesia in 3 patients where the evidence suggested neoplasm immediately below the chest wall and lateral to the sternal border Needle biopsy has been employed once (Case 18, L L), the tumor had penetrated the anterior chest wall and could be felt in the soft tissues overlying the sternum In 2 cases of superior caval obstruction (Case 17 E B, and Case 19, C B), we have employed transsternal biopsy This is a relatively simple and highly effective method of obtaining tissue without resorting to thoracotomy (Fig 8) It appears unlikely that less direct methods such as scalene node biopsy will be effective in thymic tumors as lymph node extension is not common

Surgical Excision—Ideal therapy of a neoplasm requires total removal accomplished without violation of tumor boundary and leaving no residual cell remnants Nine of the 19 patients *in* this series had surgical extirpation of their tumors, 1 died in an acute postoperative myasthenic crisis, 6 are living and apparently free of neoplasm, 2 had late recurrences of neoplasm after removal of apparently benign tumors From the technical standpoint, excision of a thymic neoplasm is usually not difficult even when there is extracapsular invasion of the fat and pericardium In 2 of the early cases, we employed midline sternotomy which is unnecessary and only adds to the morbidity and the complexity of the operation For most thymic tumors, adequate exposure is obtained through either a right or left anterior thoracotomy approach If it should be necessary, the incision can be extended by [illegible] of the

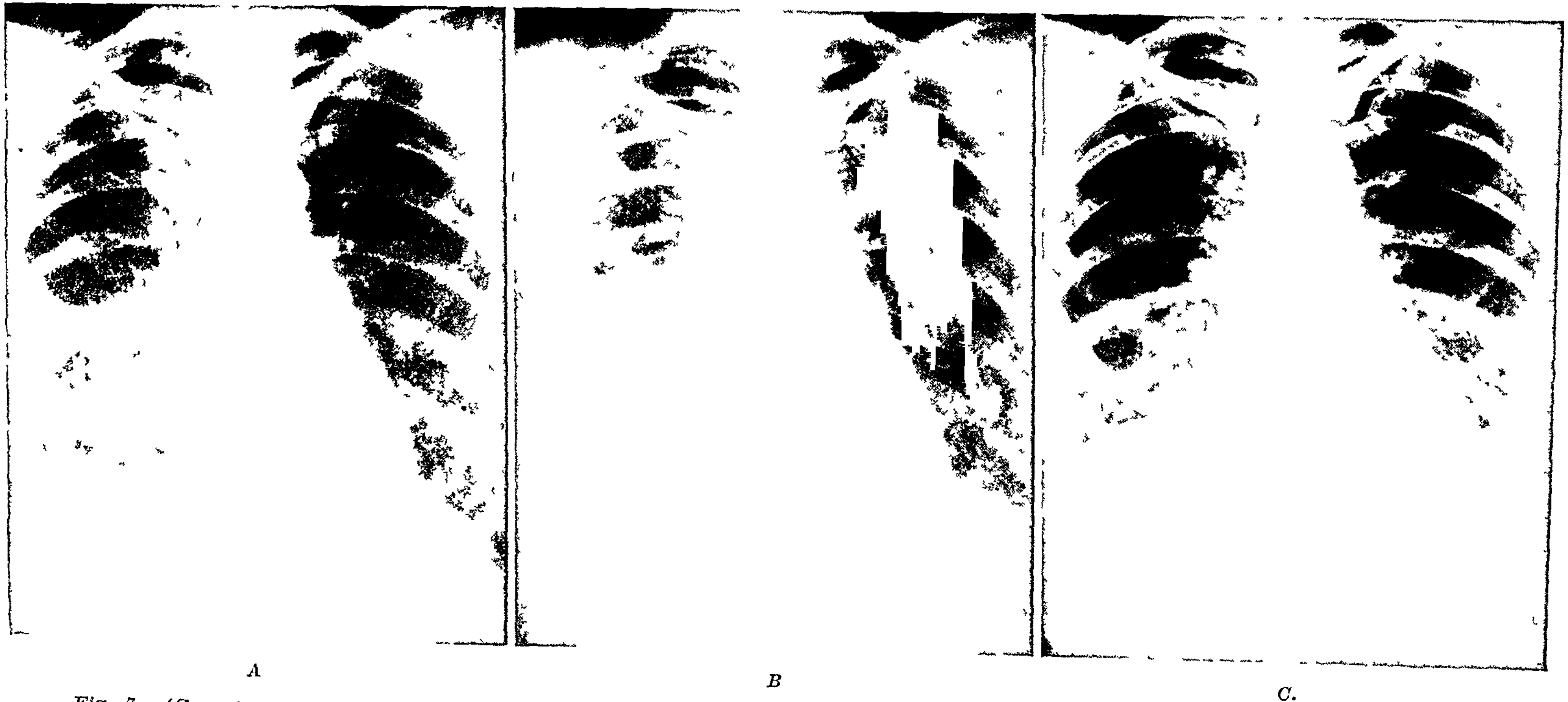
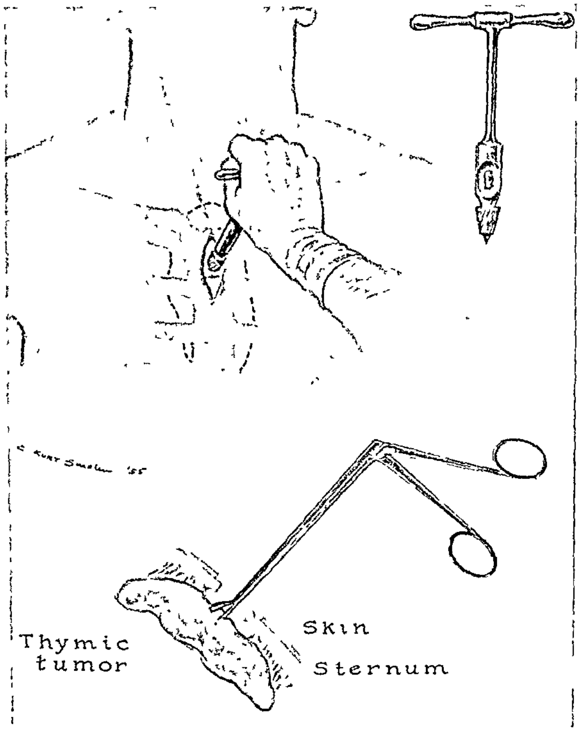


Fig 7.—(Case 1, G. L. H.) Pictorial sequence of highly malignant thymic neoplasm with pleural implantation and favorable response to radiation therapy. *A*, A reproduction of film made in February, 1951. There were no symptoms of the large anterior mediastinal tumor. A previous film date 1947, was entirely normal. *B*, Film demonstrates multiple intrapleural metastases that appeared in August, 1951. The radiographic appearance of these implants was preceded by abrupt onset of pain corresponding to phrenic distribution on the right. Thoracotomy biopsy established diagnosis of metastatic thymic carcinoma. *C*, A film made in February, 1954, following combination of radiation therapy and nitrogen mustard therapy. Shortly after this film was made, the patient died in severe myasthenic crisis. (See Table I.)

Radiation Therapy.—There is reason to believe that some neoplasms of the thymus have a tendency to regress under radiation therapy and that local implants frequently share this property. There is no way of anticipating tumor response until a full course of therapy has been given; therefore, we urge that all patients who present a nonresectable neoplasm be given a full course of radiotherapy, rather than a test dose. Of the 19 patients in our series, 10 had nonresectable neoplasms. All of these received radiation therapy with completely unpredictable results. One patient (Case 15, M. K.) is in excellent health three and one-half years after receiving 2,500 r of therapy for a highly malignant nonresectable lesion. There is little question that the majority have received benefit and prolongation of life (see Table II).



Fi. [illegible] ...tion of technique employ...
thymic [illegible] usually employed only in la...
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safe, as [illegible] the sternum and the gre...
The procedure is performed under local anesthesia

After reviewing our limited experience, since 1949, with the 19 cases presented, we believe that ideal therapy consists of surgical extirpation of the primary neoplasm followed by a full course of radiation therapy to the anterior mediastinum. It is obvious that we cannot distinguish the benign from the malignant tumor by histopathologic study. It is apparent likewise, that gross diagnosis of a benign thymic tumor is unreliable. Two of the "benign" thymomas presented metastases, two and one-half and three years after excision. We have adopted the philosophy that all thymic tumors are malignant or of malignant potentiality and consider the ideal treatment to combine excision with a full course of radiation therapy to the anterior mediastinum. The radiotherapy should begin during the immediate postoperative phase. It might well be argued that successful extirpation of the tumor would permit the surgeon to allow a period of clinical observation before employing radiation therapy. It is our contention, however, that the period of time which may elapse before late metastases appear may well go beyond the period of clinical observation and contact between surgeon and patient may be broken. This, in addition to bitter experience with 2 late recurrences in patients presumed to be cured, makes us believe that radiation therapy in the immediate postoperative phase is an integral part of therapy for all thymic neoplasms.

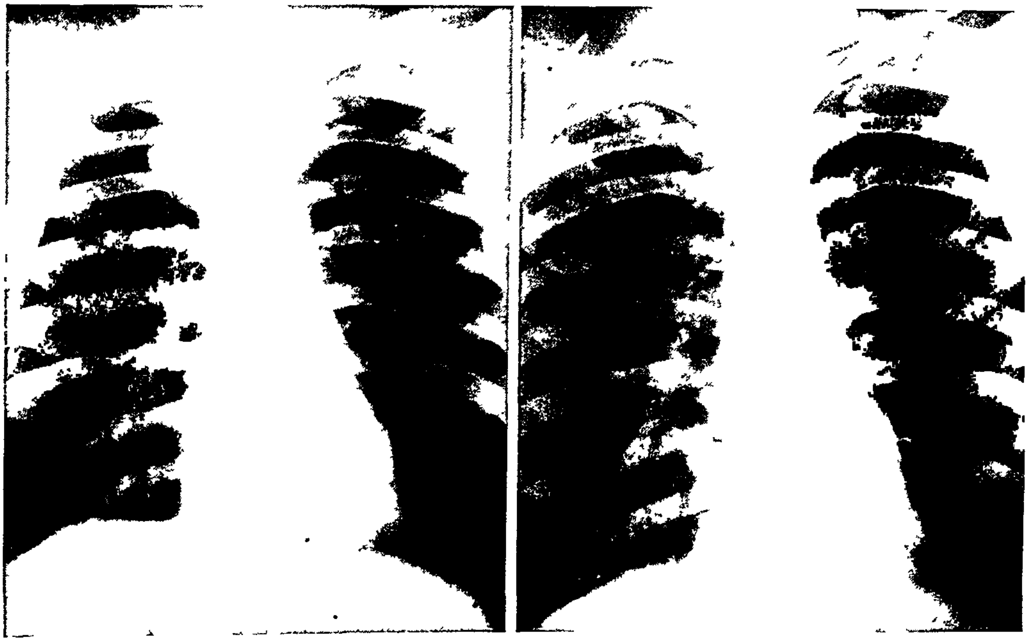

Fig. 9.—(Case 15, M. K.) *A*, Roentgenogram demonstrates malignant thymoma that was accidentally discovered during immediate postpartum. Tissue diagnosis established by limited anterior thoracotomy. *B*, Radiographic appearance of chest approximately four and one-half years after radiation therapy. The patient remains clinically well.

SUMMARY AND CONCLUSIONS

Twenty-six case histories were reviewed: 19 are believed to be thymic in origin and 7 to be intrathymic in locale, but not of truly thymic origin. The factor of malignancy predominates in this personal series, although it was not immediately apparent in every case It is our conviction that thymic tumors are malignant or potentially malignant, and that the cancer factor is of greater importance than any possible relationship with myasthenia gravis

Response to therapy is unpredictable regardless of whether the tumor is apparently benign or obviously malignant A tissue specimen is mandatory for diagnosis and may be obtained by several biopsy methods including the simple expedient of transsternal biopsy We believe that every patient who has a nonresectable thymoma should be given a full course of radiation therapy All patients who have undergone thymectomy should receive radiation therapy to the mediastinum to reduce the hazard of late recurrence

We believe that thymic neoplasms may be classified as of 2 types the lymphomatous, and the carcinomatous Employment of this classification and its variants has no value in prognosis but may improve diagnosis of primary thymic neoplasm

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DISCUSSION

DR JOHN W STRIEDER, Brookline Mass —I should like to compliment the essayists on the handling of a difficult and obscure subject and particularly on their classification I wish to call attention to two aspects one of which is well known and the other, perhaps, not so well known Dr Effler mentioned that patients with thymic malignancies may later develop myasthenia gravis We have had one such patient from whom a thymoma was removed and who two years later developed myasthenia gravis A year later, after considerable therapy, she had a total thymectomy with complete remission for two years up to this time

The occurrence of a so called benign thymoma and of a refractory anemia which failed to respond to all therapeutic measures has been observed in 2 patients at the Massachusetts Memorial Hospitals The simultaneous occurrence of two unusual pathologic states in 2 patients in a relatively brief period suggests a probable relationship between the thymic tumor and erythropoietic abnormality, a probability which was strengthened by finding several cases in the literature This unusual coincidence of abnormalities in the thymus and in the blood provides one more link in the legendary and frequently nebulous chain of evidence connecting the thymus gland with the hematopoietic system Both these patients were women aged 40 and 45, respectively, and both ultimately had excision of the thymoma without apparent effect on the course of the anemia Both have continued to run a course which has been characterized by failure to form blood At least 150 to 200 transfusions, mostly of

After reviewing our limited experience, since 1949, with the 19 cases presented, we believe that ideal therapy consists of surgical extirpation of the primary neoplasm followed by a full course of radiation therapy to the anterior mediastinum. It is obvious that we cannot distinguish the benign from the malignant tumor by histopathologic study. It is apparent likewise, that gross diagnosis of a benign thymic tumor is unreliable. Two of the "benign" thymomas presented metastases, two and one-half and three years after excision. We have adopted the philosophy that all thymic tumors are malignant or of malignant potentiality and consider the ideal treatment to combine excision with a full course of radiation therapy to the anterior mediastinum. The radiotherapy should begin during the immediate postoperative phase. It might well be argued that successful extirpation of the tumor would permit the surgeon to allow a period of clinical observation before employing radiation therapy. It is our contention, however, that the period of time which may elapse before late metastases appear may well go beyond the period of clinical observation and contact between surgeon and patient may be broken. This, in addition to bitter experience with 2 late recurrences in patients presumed to be cured, makes us believe that radiation therapy in the immediate postoperative phase is an integral part of therapy for all thymic neoplasms.

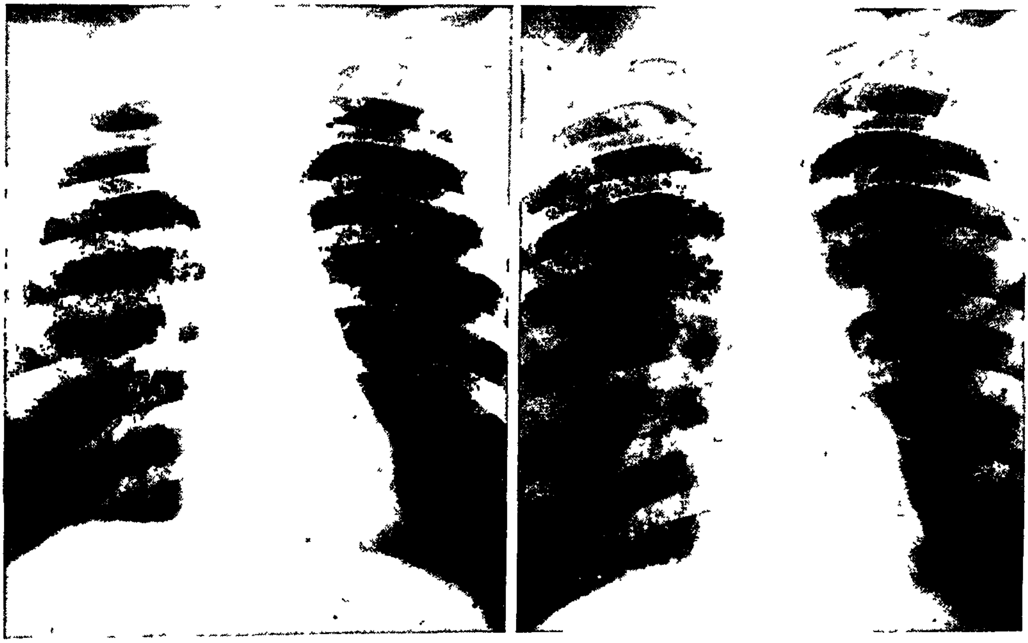

Fig. 9.—(Case 15; M.K.) *A,* Roentgenogram demonstrates malignant thymoma that was accidentally discovered during immediate postpartum. Tissue diagnosis established by limited anterior thoracotomy. *B,* Radiographic appearance of chest approximately four and one-half years after radiation therapy. The patient remains clinically well.

SUMMARY AND CONCLUSIONS

Twenty-six case histories were reviewed: 19 are believed to be thymic in origin and 7 to be intrathymic in locale, but not of truly thymic origin. The factor of malignancy predominates in this personal series, although it was not immediately apparent in every case It is our conviction that thymic tumors are malignant or potentially malignant, and that the cancer factor is of greater importance than any possible relationship with myasthenia gravis

Response to therapy is unpredictable regardless of whether the tumor is apparently benign or obviously malignant A tissue specimen is mandatory for diagnosis and may be obtained by several biopsy methods including the simple expedient of transsternal biopsy We believe that every patient who has a nonresectable thymoma should be given a full course of radiation therapy All patients who have undergone thymectomy should receive radiation therapy to the mediastinum to reduce the hazard of late recurrence

We believe that thymic neoplasms may be classified as of 2 types the lymphomatous, and the carcinomatous Employment of this classification and its variants has no value in prognosis but may improve diagnosis of primary thymic neoplasm

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DR. JOHN G. CHESNEY, Miami, Fla.—I wish to show slides of a case which I think is quite unique. This is a 21-year-old girl with no complaints, whose lesion was found on routine chest survey. This rounded lesion was found on the right side. She was operated upon and the lesion was found to be deep in the major fissure under the pleural reflection in the fissure. It lay in the anterior segment of the upper lobe and was directly related to the posterior portion of the middle lobe, and also related directly to the anterior portion of the superior segment of the lower lobe. The lesion was dissected from its bed without difficulty. It was necessary to strip off the adventitia of the lower lobe artery and what our pathologist has described as normal-appearing thymic tissue was found.

(slide) Here is a high-power section of the same tumor, which I think is unique. At least, I know of no reported ectopic thymic tissue in the lung.

DR. JAMES E. DAILEY, Houston, Tex.—I would like to report an experience with a total of 12 thymoma cases, 9 of which were benign and 3 malignant. Only 2 of this group had myasthenia gravis.

(slide) This was a 57-year-old Negro who falls into the classification Dr. Effler described, a carcinomatous type. Incidentally, this patient was operated upon in 1949 and is still alive.

(slide) The next one, a 45-year-old white man also had a carcinoma of the thymus. He died shortly after operation with a sudden massive pulmonary embolus.

(slide) This 41-year-old white woman had what the pathologist described as being in the borderline group between the noncancerous and the malignant thymoma of the type of Symmer. She died within a year from metastatic lymphosarcoma.

(slide) This next is a most interesting case. In this you will note the thin density of the tumor; it was a lipoma. The dark areas can be identified microscopically as typical thymus tissue. The diagnosis was benign lipoma of the thymus gland.

I am not sure that I would go along with Dr. Effler on x-ray therapy for *all* thymomas.

DR. HARVEY J. MENDELSOHN, Cleveland, Ohio.—I wish to compliment Dr. Effler on his excellent presentation and classification of this confusing subject of thymic tumors. There is one complication which he had occasion to encounter only once in our series of 13 cases of thymic tumors, 8 of which were malignant. This occurred in a child, aged 5 years, with a lymphosarcoma of the thymus. There was a history of six days of upper respiratory obstruction; he had been in good health otherwise until then. The problem, of course, was to treat the respiratory obstruction and to treat the tumor. Surgical incision was contraindicated and it was chosen to give him x-ray therapy. He died suddenly in thirty-six hours of respiratory obstruction after one x-ray treatment of 100 r, and before tracheotomy could be established.

Autopsy showed a lymphosarcoma surrounding the trachea and great vessels and filling the region normally occupied by the thymus.

Within a few weeks another child was admitted to the hospital with anatomically the same situation; however, he had leukemia. He had the same type of respiratory obstruction, and in this case we heeded the warning of Dr. Neuhauser of Boston and treated this child with cortisone. The tumor receded within forty-eight hours and the respiratory obstruction was relieved. I wish to mention the lympholytic effect of cortisone as an adjunct in treatment of patients who have this problem It does not always produce regression of thymic tumors or lymphomas but should be tried when respiratory obstruction is present It should be followed quickly by x ray or nitrogen mustard therapy since the cortisone effect is temporary

DR JOHN F HIGGINSON, Portland, Ore —We have 2 cases of thymic neoplasms to mention One of these typifies the multiple mistaken diagnoses mentioned by the authors as frequent This patient had a large anterior superior mediastinal mass and several other masses in the left lung field on chest x ray Bronchoscopy yielded a bronchial aspirate suspected of showing possible bronchogenic carcinoma Left pleural fluid contained only mesothelial cells Thoracotomy demonstrated multiple tumors throughout the left chest and lung and a large mass in the anterior superior mediastinum The tissue removed for biopsy was diagnosed on frozen section as metastatic bronchogenic carcinoma Later the fixed sections were felt to demonstrate a malignant thymoma Still later the pathologist changed to a diagnosis of liposarcoma, but following review of the sections throughout the country the lesion was felt to be a malignant thymoma

The second case is that of a lipoma of the thymus in a 13 year old boy This anterior mediastinal tumor weighed 589 Gm and the pathologist was explicit in stating that the tumor was a lipoma of the thymus and not a simple mediastinal lipoma encroaching upon thymic tissue No evidence of malignancy was found in this tumor

DR HOWARD K GRAY, Rochester, Minn We would certainly agree with Dr Effler and Dr McCormack that tumors of the thymus occur more frequently than has generally been appreciated Since 1940, approximately 75 patients with tumors arising from the thymus have been operated upon at the Mayo Clinic We believe thymic tumors occur even more frequently than dermoid and teratoid tumors, which are usually considered to be the most common anterior mediastinal tumors

While the relationship between myasthenia gravis and thymic tumors is poorly understood it is very real and significant It cannot be pure coincidence that thymic tumors have been found in 16 per cent of almost 500 cases of myasthenia gravis seen since 1940 or that approximately 70 per cent of all our thymic tumors have occurred in individuals with myasthenia gravis Of considerable interest, too, is the fact that we know of 5 cases in which patients who had never had myasthenia gravis previous to removal of a thymic tumor have developed typical myasthenia gravis at various intervals after removal of the thymic tumors

Whether thymic tumors should be considered as benign or malignant depends on one's definition of what constitutes benignancy and malignancy Certainly they are a unique group of tumors Microscopically they look as if they should be benign Many show cystic changes and areas of calcification and are well encapsulated and both grossly and microscopically appear to be entirely benign However, in other cases a tumor that is identical on microscopic examination will invade through the pleura and into the lung invade through the pericardium, obstruct the large veins in the mediastinum, and implant on pleural surfaces, producing a bloody pleural effusion It has been an observation of interest to us that thus far we have not seen a true thymic tumor that has metastasized or spread outside the thorax

It has been our practice to treat thymic tumors that have shown invasive characteristics with intensive deep x ray therapy postoperatively We have not used deep x ray therapy postoperatively in patients with well encapsulated tumors We have not encountered late metastases or recurrence in these apparently well localized tumors

The effect of deep x ray therapy on tumors of the thymus that could not be resected has been rather unpredictable in our experience In some it has apparently been quite effective In other cases that seemed to be identical, x ray therapy has apparently not been of much value

DR JOHN L POOL, New York, N Y There are two points I should like to make on this extremely complete presentation of the subject of thymomas In the first place, the malignancy of these lesions has been seen in the experience at Memorial Center in very much the same way as presented here today, and we agree that postoperative radiation therapy is a useful adjunct in cancer of the thymus gland, which so often has a histologic appearance similar to the lymphomatous neoplasms. The solid tumor of the thymic gland lies in the anterior mediastinal fat and it can be noted to infiltrate the perithymic fat without any clearly defined capsule. In one instance, a man in his twenties had a cystic thymic carcinoma apparently totally extirpated. The lesion was asymptomatic and found by chance. The excision was followed by radiation therapy and seven years later metastasis to the axilla was noted without any mediastinal recurrence. Another aspect of the natural life span of these neoplasms is represented by a 39-year-old woman who had myasthenic symptoms in 1950, and a chest x-ray at that time revealed an anterior mediastinal tumor with a sharply defined border, apparently 6 cm. in diameter. She was treated with Prostigmin for four and one-half years at which time repeat chest x-rays revealed the margin to be ''shaggy'' and to have a definite projection at one point. At this time she came under our care and exploratory thoracotomy revealed reticulum-cell lymphosarcoma with infiltration of adjacent lung. Malignant degeneration had apparently occurred during the intervening years. These findings reinforce to me the advisability of early excision for all undiagnosed anterior mediastinal masses.

DR. DE WITT C. DAUGHTRY, Miami, Fla.—Of our 8 cases of thymoma, 4 have been classed as malignant by our pathologists. All, except 1 patient, are alive one to eight years following surgery. The death was in a terminal case when first seen and no surgery or other therapy was recommended. One other had such extensive invasion of the mediastinum that surgical removal could not be accomplished. This was complicated by superior vena caval obstruction. Despite the extensiveness of this lesion, the patient is alive and apparently well seven years after surgery and x-ray therapy. It has been necessary to give him two courses of x-ray therapy.

Four tumors were in the usual location for thymomas while the remaining four were located elsewhere; 1 in the lower anterior mediastinum, 1 in the posterior mediastinum, and 2 within the lung. My associate, Dr. Chesney, has already mentioned one of them in some detail. The other one was cystic and located in the mid-lung field. Two of our patients complained of excessive fatigability over a period of four or five years which necessitated change in their occupation. This symptom has completely subsided in both of them since surgery.

There is no question but that thymic tumors should be explored and removed immediately upon discovery if possible. In addition, it seems wise even if complete extirpation can be accomplished to use x-ray therapy if there is any question of malignancy.

DR. J. HARVEY JOHNSTON, JR., Jackson, Miss.—Admittedly, the results of thymectomy in myasthenia gravis are unpredictable. There is increasing evidence, however, that thymectomy often effects amelioration of symptoms and occasionally cure in myasthenia gravis.

I should like to record the case of a 35-year-old white woman admitted in extremis in an acute myasthenic crisis despite 4 mg. of Prostigmin every hour by injection. Emergency tracheotomy was done which aided in evacuation of viscid tracheobronchial secretions and more effective ventilation. Despite large doses of Prostigmin and meticulous attention to tracheobronchial secretions, her condition remained critical—so much so, that all observers felt that death was inevitable with continued conservative treatment. Thus, with considerable trepidation, emergency thymectomy was performed and a well-circumscribed 8.5 by 6.5 by 3 cm. thymoma was removed and a thymic remnant was removed. The procedure was technically easy and tolerated well by the patient. The response after surgery was indeed gratifying with rapid and progressive improvement allowing return to full activity one month later on a maintenance dose of 30 mg. of oral Prostigmin per twenty-four hours.

Although this excellent result may be only a fortuitous one, it suggests that early thymectomy with the aid of tracheotomy after a short period of intensive preoperative preparation may be lifesaving in severe cases of myasthenic crisis.

DR EDGAR W DAVIS, Washington, D C—We too have been quite disturbed by the confusion and uncertainty about thymic neoplasia In the brief time available I would like particularly to stress the too little appreciated tendency toward cyst formation Small cysts which are so typical of thymomas cause no diagnostic difficulty, but when the cystic element predominates, the thymic origin of the tumor may be completely missed Actually these "thymic cysts" have received scant attention in the literature, so much so that perhaps the most famous such specimen, namely the one removed by Dr Blalock in his first thymectomy for myasthenia gravis, has been overlooked in all recent reviews on the subject We seriously question whether such cysts are as rare as the literature suggests in view of 5 thymic cysts among our personal series of 17 thymoma cases

(Slide) This is the chest x ray of a 10 year old boy This mediastinal mass had been discovered in a fortuitous roentgenogram taken several years earlier, and because of the likelihood of its representing an enlarged thymus gland, he had received small amounts of x ray therapy Ultimately, however, its increasing size prompted its resection Grossly, it was a thin walled multilocular cyst containing clear fluid The significance of the thymic tissue throughout the septa was appreciated only recently when the sections were re examined

(Slide) This 24 year old medical student presented a comparable negative history and an identical lesion

(Slide) This is the x ray of a 35 year old woman who like the others, had no symptoms (Slide) It was a unilocular cyst filled with brownish fluid containing many glittering yellow particles The thymic stroma in its wall was interpreted originally as a degenerating lymphosarcoma No irradiation was given and she remains well seven and one half years later

(Slide) Again a young woman who had no complaints The preoperative diagnosis was thymoma (Slide) The specimen was a remarkable, unilocular cyst located in the left lobe of the thymus, the entire gland being seen here (Slide) On section it was found to contain thick grumous material like that of any cystic teratoid tumor No doubt it might have been mistaken for a dermoid cyst had not the entire thymus been resected and its relationship thus preserved

(Slide) This last case, in a middle aged man more or less bridges the gap between thymic and dermoid cysts, for the lesion was a classical dermoid cyst containing hair The wall was comprised of thymic tissue including Hassall's corpuscles Obviously, a case of this sort goes a long way toward confirming a more than casual relationship between teratoid tumors of the mediastinum and the thymus gland

DR PHILIP W SMITH, Marion, Ohio—We have studied 32 cases of primary thymic neoplasms of the type described by Dr Effler These cases were from the Armed Forces Institute of Pathology and from the University of Michigan Twelve of these cases had associated myasthenia gravis 3 had associated Cushing's disease and 17 had no apparent associated neuromuscular or endocrine disorder Histologically the neoplasms in all three groups were indistinguishable They are not to be confused with the true lymphomas which may involve the thymus, though at times their histologic morphology is similar enough to make differentiation difficult A more simplified classification of these neoplasms seems warranted in the light of present knowledge We believe that the neoplasms in all of the groups described by Dr Effler have a common cell of origin, the so called thymic reticulum cell or, perhaps more properly, the thymic syncytial cell These tumors present a variety of histologic appearances both between different tumors and in different areas of the same tumor It is probably superfluous to subclassify them histologically according to their predominant cell pattern because it tells us nothing of their biologic behavior

We agree that all thymic neoplasms should be considered as actually or potentially malignant The criteria for malignancy must usually depend on their gross characteristics and upon clinical evaluation of their biologic behavior rather than on histopathologic evidence Three of these 32 tumors did show regional embolic lymph node metastases Nearly all showed capsular invasion, and extensive local infiltration and implantation were common

A prompt tissue diagnosis certainly is essential; and, if possible, total excision of these neoplasms with any remaining thymus should be accomplished. If total excision is not possible, the mediastinal structures often may be mechanically decompressed by removal of a portion of the tumor. Local implantation with appropriate radioactive materials such as iridium and/or externally applied roentgen therapy is definitely indicated if one is not sure that excision is complete. The response of these tumors to irradiation is variable but significantly worth while in a good percentage of them.

Finally, for the sake of the record, the photomicrograph just shown by Dr. Chesney I believe is misclassified as a thymic neoplasm occurring in the lung. A similar tumor is to be found in the files of the Armed Forces Institute of Pathology, also occurring in the lung. I do not believe it is related to the thymus at all but is actually a type of lymphoma. The epithelial-appearing cell clusters are not Hassall's corpuscles but collections of endothelial cells.

DR. DONALD B. EFFLER (Closing).—I want to thank the discussants, and to thank Dr. Meade for closing the discussion before someone less friendly than the others had a chance to speak. I would like to close with the summary which appears in the paper by simply stating that we believe that indications for surgical intervention are the same as in all mediastinal tumors and contraindications are perhaps the same. Tissue diagnosis is mandatory and in all patients, regardless of whether or not the tumor can be removed, a full course of postoperative radiation therapy is recommended.

PULMONARY RESECTION IN ACTIVE CAVITARY (OPEN-POSITIVE) TUBERCULOSIS

ROBERT H. HOLLAND, M.D.* (BY INVITATION), SUNMOUNT, N. Y., JOHN W. BELL, M.D. (BY INVITATION), SUNMOUNT, N. Y., AND EDWARD S. WELLES, M.D., SARANAC LAKE, N. Y.

CURRENT attempts to control cavitary tuberculosis with prolonged chemotherapy result in a predictable incidence of medical failures. For example (Fig. 1), there were no definitive surgical procedures in this random group of 179 original treatment cavitary cases during the first 12 months of chemotherapy with streptomycin and PAS. Following 6 months of drug therapy, 29 per cent had residual cavities and positive sputum, and after 12 months, 18 per cent still had positive sputum and persistent cavities.

In the majority of recent reports,[1-3] no attempt has been made to correlate the preoperative susceptibility of the tubercle bacilli with the postresection complications and therapeutic results. Falk and Tucker[4] state that bacterial resistance to the chemotherapeutic agents at the time of surgery appeared to have little effect upon relapse or failure in their series of resections. Differing from this opinion, we have found this information essential to the proper management of these cases and directly related to the complications and end results.

MATERIAL

This study is a review of 83 sections in 75 patients who had positive sputum and persistent cavities at the time of operation. For convenience in classification, as suggested by Decker, Raleigh, and Welles,[5] the term "open" signifies the persistence of an annular shadow, recognized as such on planigrams. The report of at least one positive sputum culture within the three-month period immediately prior to surgery is sufficient to designate the case as "positive."

Original chemotherapy cases are those in which treatment was started on the first admission, or if on readmission or transfer, not more than 6 months of drug therapy with no interruptions over 2 weeks. All other patients, because of unknown intervals between drug regimens, are considered to be in the re-treatment group.

Further subdivision of the original and re-treatment groups is based on the susceptibility pattern of the tubercle bacilli in the patient, both on the

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From the Veterans Administration Hospital, Sunmount, N. Y.

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 24 to 26, 1955.

*Present address: Veterans Administration Hospital, Dallas 2, Texas.

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*Present address: Veterans Administration Hospital, Dallas 2, Texas admission sputum culture and on the last positive specimen before operation. Susceptible tubercle bacilli are those showing less growth, than in controls, in at least 2 of the 3 tubes of ATS medium containing 15 μg of streptomycin, 10 μg of PAS, and 1 μg of INH per milliliter.

TABLE I. ADMISSION BACTERIAL SUSCEPTIBILITIES

Original Chemotherapy.—	
Susceptible	28
Resistant	0
Re-treatment.—	
Susceptible	42
Resistant	13
	83

On admission (Table I), there were 28 original chemotherapy-susceptible cases, and none were found to be resistant. Forty-two were considered as re-treatment susceptible, and 13 showed resistant bacilli at the beginning of re-treatment. Drug therapy ranged from 2 to 32 months preoperatively, being quite prolonged early in the series in an attempt to convert the sputum.

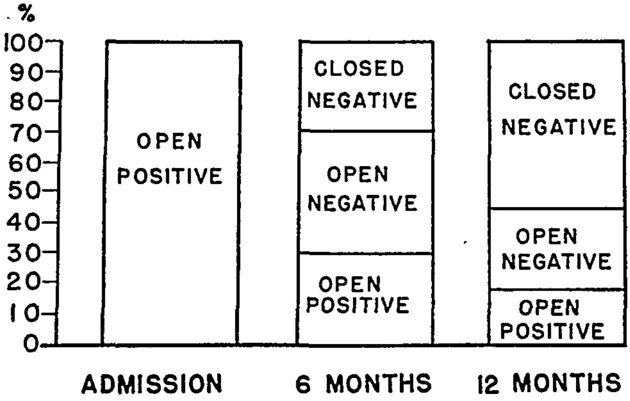

Fig. 1.—Sputum conversion and cavity closure in original chemotherapy.

At the time of operation (Table II), 19 original treatment patients still had susceptible organisms while in 9 the bacilli had become completely resistant. Of the 42 patients re-treated as susceptible, 19 had developed resistant bacilli by the time they were considered for resection of surgery. Thus, a total of 32 patients came to operation in the re-treatment resistant state.

TABLE II. PREOPERATIVE BACTERIAL SUSCEPTIBILITIES

		Net (Change)
Original Chemotherapy.—		
Susceptible	19	↓
Resistant	9	9
Re-treatment Chemotherapy.—		
Susceptible	23	↓
Resistant	32	19

OPERATIVE PROCEDURES

The operations were usually formidable. Nineteen patients had preresection thoracoplasties, 4 had permanent phrenic damage, 2 had Monaldi procedures, 19 had pneumothorax, and 11 were classical thoracoplasty failures. All of these procedures involved the resected side, making the operation more difficult technically, and the postoperative course more complicated physiologically. Wedge resection was performed only twice as a solitary procedure; segmental resection, 44 times; lobectomy, 34 times; and pneumonectomy, 3 times. There were no operative deaths, that is, within 60 days following surgery.

COMPLICATIONS

Major complications of tuberculosis which are defined as bronchopleural fistula, empyema, spread or reactivation of disease, and chest wall infections, are correlated with respect to preoperative susceptibilities of the bacilli in the patient (Fig. 2). In the original chemotherapy-susceptible group, there was 1 major complication in 19 resections, and, in the resistant group, 5 in 9 resections. In the re-treatment susceptible group, there were 3 major complications in 22 resections, and, in the resistant group, 9 in 33 resections. Nine patients in the resistant groups had viomycin and Terramycin added to their chemotherapy protocol 6 weeks prior to operation and were continued on this program at least 8 weeks postoperatively. In this group there were 3 major complications in 9 resections.

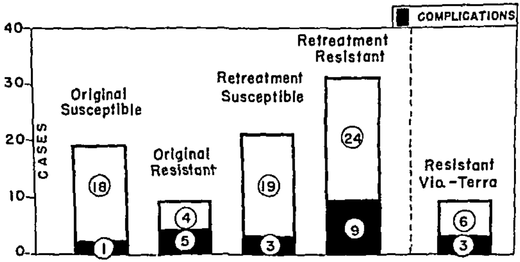

Fig. 2—Major complications

THERAPEUTIC RESULTS

Complete follow-up studies on 72 of these 75 patients range from 3 to 52 months, and are again classified in relation to the preoperative bacterial susceptibilities (Table III). Activity is defined as either radiologic or bacteriologic instability within 3 months of this review. In the original treatment-susceptible group, no cases are active, 19 are inactive, and all patients are living; and in the resistant group, 1 case is active, 5 are inactive, and 3 patients are dead. In the re-treatment-susceptible group, 1 case is active, 23 are inactive, and 1 patient died of carcinoma of the tongue with metastases; and in the resistant group, 6 are active, 22 are inactive, and 1 patient is dead.

Of the 9 patients in the resistant groups treated with viomycin and Terramycin, 4 have active cases, 4 inactive, and 1 is dead. Therefore, of the 72 patients followed, 8 have active cases, 64 inactive, and 4 died of tuberculosis which makes a satisfactory arrest rate of 95.5 per cent in the susceptible groups, and 71 per cent in the resistant groups.

TABLE III. CURRENT DISEASE STATUS

	ACTIVE	INACTIVE	DEAD
Original Chemotherapy.—			
Susceptible	0	14	0
Resistant	1	5	3
Re-treatment Chemotherapy.—			
Susceptible	1	18	1*
Resistant	6	22	1
Resistant Viomycin Terramycin	4	4	1

*Died of cancer of the tongue with metastases.

DISCUSSION

Certain radiologic and bacteriologic findings, which are presented below, have been extremely useful in anticipating medical failures during the early months of chemotherapy.

Radiologic.—

1. Cavity size: 3 cm. or over.

2. Cavity location: (a) extreme apical or posterior; (b) those persisting under a thoracoplasty (Fig. 3, *a*).

3. Cavity configuration: (a) thick-walled, and (b) hanging (Fig. 3, *b*); and (c) stellate (Fig. 3, *c*).

4. Cavity number: multiple adjacent, or those with a honeycomb appearance (Fig. 3, *d*).

5. Cavity with associated bronchial abnormalities: (a) stenosis; (b) ectasia.

Bacteriologic.—

1. Failure to reduce the bacillary content of the sputum in the first 3 months of chemotherapy.

2. Development of partial resistance to streptomycin, PAS, or INH.

3. Failure to convert the sputum in the first 6 months of chemotherapy.

When one or more of these radiologic findings is present, the cavity will usually not close, and when combined with one or more of the bacteriologic findings, sputum conversion is unlikely.

In the candidate for resection who is considered a predictable medical failure, there is little to be gained by prolonging treatment to the point where bacterial resistance emerges. Although the achievement of sputum conversion is desirable, even the open negative state does not guarantee the presence of susceptible bacilli.[6]

Vigilance is particularly necessary in the re-treatment group where the rapid emergence of drug resistance is a common phenomenon.[7] These patients should be considered for resection several months earlier than their original chemotherapy counterparts and appropriate changes made in their medical protocol if resistance is developing.

The patient with uncertain drug coverage, or known resistance, should be bronchoscoped prior to resection In these patients, transection of unrecognized endobronchial disease or spillage of tuberculous material at operation may invite pleural complications The plan at operation in the resistant patient is to perform a wide, clean resection, which usually means a lobectomy,

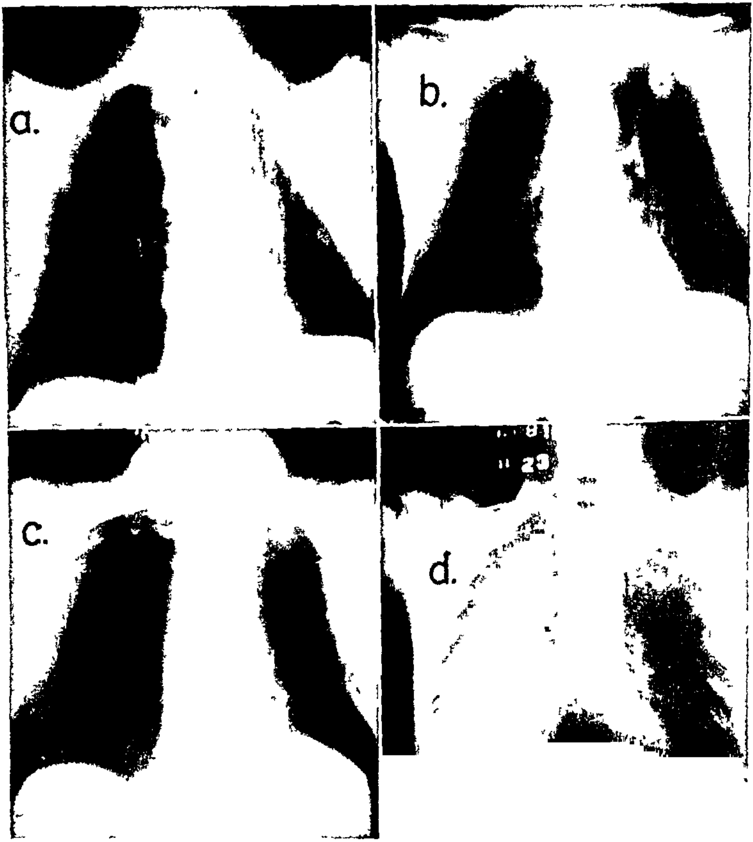

Fig 3—Planigrams illustrating various types of cavities that antituberculosis chemotherapy is not likely to close (*a*) cavity persisting under a thoracoplasty (*b*) thick-walled and hanging cavity, (*c*) stellate cavity, and (*d*) multiple adjacent cavities

unless the disease is extremely well circumscribed. This is in contrast to the practice followed in the susceptible group where limited resections have been found to be perfectly safe.

Murphy[8] reports satisfactory results in only 77.6 per cent of his operations for cavitary resection, as compared to 94.7 per cent in those operations where closed lesions were resected. In resections where the sputum was positive, 82.8 per cent of the patients had good results, as compared with 94.4 per cent where it was negative. These statistics are strikingly similar to ours when the bacterial susceptibility data are omitted. Of the three factors, open cavity, positive sputum, and bacterial resistance, we think the latter is the most significant in regard to the postresection complications and therapeutic results.

SUMMARY

1. Eighty-three resections in 75 patients with residual cavities and positive sputum are reviewed. An attempt is made to correlate the preoperative findings on tubercule bacilli susceptibility with the complications and therapeutic results.
2. In the original treatment-susceptible patient, resection should probably be performed between the third and sixth month of chemotherapy, and, in the re-treatment patient, several months earlier in order to avoid the development of bacterial resistance.
3. Of the three factors, open cavity, positive sputum, and bacterial resistance, we believe the latter to be the most significant in regard to the postresection complications and therapeutic results.
4. Despite the presence of open cavity and positive sputum, we have had excellent surgical results in our susceptible patients, and only fair results in those who were resistant.
5. The addition of viomycin and Terramycin to the chemotherapy protocol of 9 resistant patients, 6 weeks preoperatively and continued postoperatively, did not seem to alter the end result.

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DISCUSSION

DR. WATTS R. WEBB, Sanatorium, Miss.—I want to congratulate this group on their excellent presentation and certainly on some very excellent results in handling a very difficult situation. We have attempted to avoid development of this status, at least in the original treatment group, by the utilization of various teams of the drugs in order that we might always have a group of drugs to which the bacteria presumably would not be resistant. That is, ordinarily we start out with streptomycin and PAS and later on when there appears some evidence of resistance we switch over to isoniazid and Promizole and, later on, as some of the other antibiotics have been developed, we use still a different team. We have on occasion used as many as three teams of antibiotics on a single patient. We feel that this has given us, at least at the time of operation, a team that was effective against the bacteria present at that particular time. In contradistinction to their results we have found that we were thus able to continue effective drug treatment until these patients, judging by the appearance of the contralateral side and other factors, were ready for surgery. Our results in the group that have been treated with various teams of drugs have been superior to those in the group where operation was performed in the pesence of bacterial resistance.

We do concur that an earlier attack on the cavitary lesion is indicated whenever the condition of the remaining lung permits.

DR. WILLIAM M. LEES, Chicago, Ill.—It is encouraging to note that people in the East are also seeing some of these far-advanced and moderately advanced cases that we are seeing in Chicago. This is especially true in view of the fact that a review of the data as recently published in that illustrious journal, the *Chicago Tribune*, has revealed that in some areas of the country the primary indication for surgery is the fact that there are a few flyspecks on the x-ray and an occasional positive sputum; whereas in other parts of the country the large cavities, openly revealed, are permitted to remain, just because the sputum is negative.

In reviewing more than 300 resections during the last 2 years or so, I would like to confirm some of the observations reported by Dr. Holland in that more than 85 per cent of our complications have occurred in those patients with preoperative sputa positive. The majority of these patients have had intermittent chemotherapy, to be sure. However, a number have had continuous chemotherapy, and I think we will find that it is in this group that we are going to encounter most of the complications, at least, from the point of view of the pathology present.

In this group, I am referring to the pathologic specimens which, when studied carefully, will in practically all instances reveal the presence of tuberculous endobronchial disease, not necessarily at the bronchocavitary junction, but at some distance from this area. I believe this plays a considerable role in postoperative complications. We, too, have found that the complication rate is higher in those who have tubercle bacilli resistant to all the drugs. We also attempt to keep viomycin in reserve and we hope that perhaps seromycin, when it becomes available, may be of some benefit in this rather tragic group, small though it is.

DR. FELIX A. HUGHES, JR., Memphis, Tenn.—Thanks to Dr. Barnwell, I have the results of a study of morphology and bacteriology of resected tuberculous lesions in the VA-Army-Navy cooperative study. In my opinion, the data may be said to agree with the recommendation for early surgery. These patients, from 35 hospitals, had operations performed under the supervision of members of this Association.

(slide) This slide shows the percentage of negative lesions, related to the months of chemotherapy treatment, in 494 patients with closed lesions who were clinically negative for acid-fast bacilli. No patients were selected for study with less than 4 months of treatment. Here you can see the bacteriology of the lesions changed very little after 4 to 5 months of chemotherapy. What gain is obtained is accompanied by a gain in positive smears even after 12 months of therapy.

(slide) This slide relates closed lesion bacteriology and chemotherapy regimen to how many months the patients were clinically negative. A gram a day of streptomycin plus PAS administered until the patient was clinically negative for 2 months resulted in excised lesions that showed about as high a percentage of negativity as was found at the end of a year. This confirms, I think, the judgment of the people who resected these lesions, because the lesions showed such a high negative percentage, but it also confirms the observation that as soon as the patient is clinically ready, the resection should be carried out. Even with streptomycin twice a week plus daily PAS, apparently the maximum effect on the lesion is reached after the patient has been clinically negative for 4 to 6 months.

(slide) When considering only patients who had lesions with negative cultures but positive smears, which I think indicates less therapeutic effect than a negative culture with a negative smear, a gram of streptomycin plus PAS daily caused a maximum improvement in the lesion, of being clinically negative, at the end of four months. With streptomycin twice a week plus daily PAS, there is a slight therapeutic increase throughout the 12-month period.

(slide) When it comes to resected open lesions, although still negative clinically, there is a marked decrease in the percentage of lesions that are negative during the period between 4 and 5 months and 6 and 7 months. I think this is very significant and is why the VA studies confirm the recommendations given by Drs. Holland, Bell, and Welles.

This is hindsight evidence, but indicates that astute clinical judgment in the handling of tuberculosis cases is more indicated than ever. From my experience with the resection of approximately 500 tuberculous lesions, I also agree with the recommendations presented here. Our findings have agreed too that when the tubercle bacilli are resistant there is a tremendous increase in the percentage of complications. When there is a predictable medical failure, which frequently can be determined early, resection in suitable patients should be done at the earliest optimum time.

DR. WINFIELD O. KELLEY, Norwich, Conn.—Our findings in the tuberculous cavity are similar to those of Dr. Holland. I would like to stress a point which we feel is very important in evaluating these cases preoperatively. In going through our series of the last 243 cases, I was able to pick out 167 with cavities which perhaps agree with the principles followed by Dr. Holland in his classification. It seems that we are unable to evaluate the cavity situation by regular x-rays alone and we have come to rely upon laminagrams almost exclusively. It has been difficult also, even in that respect because of the interpretation of the roentgenologist and we found we had to include probable cavities even when we used laminagraphic interpretation.

(slide) There were 167 specimens from patients with cavitation; 121 showed acid-fast bacilli by smear or culture, making 72 per cent. There were 104 with a positive smear, at least one positive smear; 90 had positive cultures and only 70 were positive both by smear and culture.

(slide) This may be a more rigid classification than was used by Dr. Holland and his group. For open cavity cases, we specify that the sputum had to be positive within 6 weeks of operation; there was laminagraphic evidence of cavity preoperatively and there was proof that there was a cavity with a positive finding of bacilli in the cavity that had been resected. In this series of 167 specimens from patients with cavitation, there were 121 specimens (72 per cent) which were positive for acid-fast bacilli by smear or culture; 104 (62 per cent) which were positive for acid-fast bacilli at least by smear; 90 (54 per cent) which were positive for acid-fast bacilli at least by culture; and 70 (42 per cent) which were positive for acid-fast bacilli by smear and culture.

The results in these cases were as follows: There were 24 patients whose tests were still positive by smear or culture postoperatively but in 2 of these the organisms proved to be saprophytes. Then we had 22 cases that had to be further evaluated. Most of the specimens became negative soon afterward, the time varying between 1 and 3 months.

Three patients died, not early deaths but late deaths, one of bacterial endocarditis [illegible] with advanced tuberculosis, another had far advanced tuberculosis, the third [illegible] accidental death One of these patients had a relapse and [illegible] in the hospital

(slide) In order to point out some of the problems in evaluating these cases [illegible] shows the x ray after 10 months of streptomycin and PAS therapy The sputum was positive on admission but became negative and was negative at the time of operation There is a small lesion in the left lung which is probably important as most of you can see (slide) This is the lesion which was resected

(slide) There is an obvious cavity here of rather large size, and another smaller lesion here This patient's specimen was negative by smear but was positive by culture This case could not be used in our series even though it was proved to be a positive case

DR NORMAN J WILSON, Brookline, Mass—I should like to compliment Dr Holland, Dr Bell, and Dr Welles, not only for calling our attention to a timely and important subject, but also for calling attention to other important criteria which determine the success of surgical treatment other than those related to drug therapy We have heard a great deal in the literature, and also here this morning, about drug criteria and the fact that a positive sputum and open cavity are sources of danger to the patient undergoing resection

I should like to ask, do you really think that is true? Or is it the fact that most patients of this type are those who had the most serious tuberculosis to begin with and those who represent treatment failures? Many patients with open cavities who have been treated over long periods of time, and especially with recurring or persisting positive sputum, have partial to complete resistance This fact would seem to be of great importance compared with the fact that a positive sputum or an open cavity was present

Recently Dr Edward Hayes, Dr Overholt, and I made a study of a group of patients on their first course of drugs, who had been treated by resection during the early months of treatment One hundred and eight had resection performed with less than a month of drug therapy The only difference in statistics in the groups that were studied was that the earlier the resection was done the better the results, provided the surgical criteria were abided by

What are the surgical criteria? First that the only threatening lesion is within the unit of lung to be resected and that all other lesions have been controlled by drug therapy and medical treatment Second, that the patient have an adequate respiratory reserve Third, that the patient has had intelligent drug treatment sufficient to eliminate toxic symptoms and excessive sputum It is our experience that when these fundamental surgical criteria are present, the duration of preoperative chemotherapy is unrelated to the results, except that those patients who are operated on very early during the course of drug treatment have slightly better results than those operated on later

One other thing I would like to pass on We have found that pyrazinamide is the best drug to protect the patients in serious condition who have already had prolonged chemotherapy while undergoing resection

DR A M DECKER, Richmond, Va—I feel an obligation to make two points for the record First is a point of which Dr Holland and his colleagues are fully aware, but I am afraid they will perhaps not have time to clarify it directly, and I feel that some of the data might be misinterpreted The first slide that was presented showed a sharp increase in the per cent of conversion and cavity closure which occurred between 6 and 12 months Those percentages are correct However, this is a tapering study with some failures having been removed from the study after 6 months by various means of surgical intervention So actually the percentage of cavity closure and percentage of failure in the over all group does not change greatly after 6 months The other point I would like to make is that we are not yet certain of the exact level of significance of drug resistance I speak specifically of streptomycin I think that resistance to [illegible] µg of streptomycin per cubic centimeter does not correlate with clinical drug resistance as well as resistance to 15 µg There is certainly a place for further st[illegible] along th[illegible]

DR. BELL (Closing).—We are very glad to have had the opportunity of shifting the spotlight to tuberculosis, a disease which the lay press and possibly some physicians seem to believe is no longer a medical problem. Despite the use of all available antituberculosis drugs and modern resection technique, we have been unable to eliminate this disease. You have just heard the morbidity of this chronic relapsing disease. It is still present, and Dr. Medlar has predicted that it will probably increase in coming years.

With regard to Dr. Webb's comment on the various drug combinations by which they have tried to convert these cases to the negative-sputum state, our patients have had a variety of drug combinations, most of which have been continued to the point of resistance rather fruitlessly. In fact, patients who were resistant to streptomycin and PAS will, when isoniazid is added, in effect be getting INH alone and rapidly become resistant to it; thus using one of the best drugs left for surgical coverage. We have not had a great deal of experience with viomycin and Terramycin and Pyrazinamide but as you have seen, the results with viomycin are not encouraging. The number in our series is not great and we obviously do not present this material because we are particularly proud of the results. Nor do we wish to imply that the unsatisfactory cases are necessarily surgical failures; or even just chemotherapeutic failures. Actually, they are probably total failures of both medical and surgical treatment.

We should like to emphasize one point in the current resection surgery of tuberculosis. This is the important consideration of whether or not the surgical candidate is harboring resistant tubercle bacilli. We have just mentioned that late tuberculosis complications occurred in one third of the patients with resistant bacilli who had resections. This incidence is reminiscent of the forbidding morbidity whch occurred in the predrug era. The problem of resistant bacilli is not confined to patients with cavitary disease and positive sputum. We have become aware that even the patient who comes for resection with sputum negative for 3 months or more may harbor, in 10 to 15 per cent of the cases, resistant tubercle bacilli which simply are not being sloughed into the bronchial tree.

In 500 resections at Sunmount Veterans Hospital since 1950, we have had 17 cases of postoperative parenchymal reactivation which have followed at varying intervals after resection. Of these patients, 13 had negative sputum at the time of operation. Two had no cavities, and 11 were operated upon because of the open negative state. The remaining 4 had open cavities and positive sputum. Only 2 of the 17 postresection tuberculosis reactivation cases had susceptible tubercle bacilli. In other words, 15 were resistant. In 10 of the 17 patients, all of whom had both x-ray and bacteriologic relapse, all palpable disease had been removed. The microscopic spillage, which probably occurs in nearly 100 per cent of resections, is apparently innocuous in the patient with good drug control but is not safe in the resistant patient. Segmental resections or wedge resections which open raw surfaces may clear disease by only narrow margins and may also result in transection of unrecognized endobronchial disease at a subsegmental level, as mentioned by Dr. Lees. In view of these facts, we have favored lobectomy in any patient who is suspected of harboring resistant bacilli and who has uncertain drug control, regardless of whether or not the sputum was positive. In 8 patients in whom resistance was not suspected, parenchymal reactivation followed very promptly after segmental excision. Secondary lobectomies were necessary and in 1 case pneumonectomy became necessary to control the disease.

We should like to thank the discussants.

SIMULTANEOUS BILATERAL RESECTION FOR PULMONARY TUBERCULOSIS IN MENTAL PATIENTS

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ANOKA AND MINNEAPOLIS, MINN.

INTRODUCTION

WHEN in any field of surgery a single operation can successfully replace two operations, an advance has been made. In abdominal surgery staged resections of the colon have been abandoned except when there is obstruction. Only at the beginning were two operations rather than one needed to resect cancer of the pancreas. Excision of cancer of the middle third of the esophagus through the right chest apparently required a two-stage operation at first[14] but it no longer does. It is a surprising anachronism to find recently that a two-stage operation is still recommended for pharyngo-esophageal diverticula[11]; most surgeons gave it up years ago. More examples could be added but the point is clear. One operation is better than two provided, of course, that it accomplishes as much with equivalent risk and disability. This paper describes one way in which a single operative procedure can replace a two-staged operation in the surgery of pulmonary tuberculosis.

Pulmonary tuberculosis frequently spreads bilaterally[15] and it may progress far enough so that a bilateral resection is needed. Conventionally, the disease is excised at two separate operations through two long posterolateral incisions. This is not necessary, for, due to the anatomic distribution of the disease, it is technically quite easy to resect both sides through a single incision. Usually tuberculosis infects the superior poles of the lung and this region, especially, may be reached on both sides through a single, transverse, anterior incision.

It is not difficult to find reasons that favor a simultaneous rather than a staged bilateral resection for selected patients with bilateral disease. The total time of treatment and disability could be shortened and the expense greatly reduced with a single operation. The danger of postoperative aspiration or spread from residual disease is gone if this disease has been taken at the first operation. Then too, respiratory stress may actually be less after a simultaneous bilateral operation than after the second stage of a staged procedure.

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Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 24 to 26, 1955.

This reasoning might be applied to some of the patients in any tuberculosis sanatorium. Actually, the simultaneous bilateral operation has been done only on the inmates of the Anoka State Mental Hospital and for these particular patients there has been, in our experience, at least one special reason favoring the use of a simultaneous bilateral operation. At the time of writing, 94 patients in the Tuberculosis Unit of the Anoka State Mental Hospital are awaiting surgery. For each of them surgery has been recommended by a joint medical-surgical conference and they are ready, but operating time and facilities are limited and they must wait. With an overwhelming surgical load such as this, a reasonable method of accelerating the care of some patients, such as the simultaneous bilateral resection, seems fully justified.

This long list of patients awaiting surgery reflects merely one facet of a trying problem—tuberculosis in state mental hospitals. The high incidence of the disease in this population is well recognized.[2, 5] The problem has not been solved but at least the management of this situation has been progressive in Minnesota. All mental patients with tuberculosis are isolated in one modern unit which is part of a larger mental hospital. The plan of control and treatment, as well as our experience with resectional surgery in these patients, has been described in another publication.[12] Mental patients with tuberculosis deserve modern care in their own right as ill human beings but they must have it for another reason too. As an important remaining "seed bed" of the disease, they menace the public health.

The history of simultaneous bilateral pulmonary resections is meager. Some possibly apocryphal evidence suggests that one of the first pulmonary resections attempted was actually a bilateral operation done by Block in 1882.[23] It failed. The only medical report, at the time, about this operation was by a correspondent to the *Boston Medical and Surgical Journal* who had not seen the operation.[24] Block himself apparently did not find time to record the case, probably because he was incapacitated with the turmoil that led to his suicide—a direct consequence of his failure. Thus, the operation's history began on a melancholy note.

In recent times Overholt[18] has preceded us. In each of three reported cases, he used a bilateral posterolateral incision to resect segments of both lungs. The disease was bronchiectasis in two cases and the third case was mixed—a tuberculoma was found on one side and a hamartoma on the other. Our first report on the simultaneous bilateral operation was made in 1954.[22]

INDICATIONS

Rather than depend upon antibiotics alone[16] we have preferred, whenever possible, to resect even residual necrotic foci as well as inspissated cavities and more advanced disease.[1, 4, 21] All types of resectional surgery have been used liberally on mental patients with tuberculosis, perhaps even more liberally than resection would have been used for the same stages of disease in sane patients. We have tried to bring each patient to a state of health where he could be removed from the tuberculosis section and returned to the crowded general wards of the mental hospital. This transfer requires caution. In a state mental hospital tuberculosis is liable to be contagious. *Even an infrequent positive sputum arising from an apparently inactive disease can be dangerous if it is produced by an irresponsible patient.* Before they leave isolation, patients must show consistently negative gastric cultures and x-rays with no evidence of tuberculosis or, at most, only minimal scarring. *In general,* then, resection has been recommended for all patients with stable, fibronodular or more advanced disease if the disease appeared to be resectable without undue risk.

The simultaneous bilateral operation has been used for a select fraction of those needing surgery. We have chosen ambulatory patients in relatively good physical health who seemed to require no more than segmental resections on each side. In most cases the gastric cultures were negative and the roentgenologic appearance had been stable for some time. Young patients were preferred. As a matter of fact, however, the patients who had the simultaneous bilateral procedure averaged about the same age as the other surgical patients. No particular psychotic state has contraindicated the operation.

PREOPERATIVE PREPARATION

Preoperative management did not differ from that which preceded unilateral resections. The patients received antibiotics for an average of 12 months (3 to 35 months) before surgery. All had 0.5 Gm. of dihydrostreptomycin twice a week combined with 200 mg. of isoniazid daily. Five patients also took para-aminosalicylic acid during part of their course. Bed rest has proved impractical for most patients with mental disease and so it was not used.

TECHNIQUE OF THE SIMULTANEOUS BILATERAL RESECTION

A Pentothal-curare mixture is used for anesthetic induction and endotracheal intubation with the subsequent addition of nitrous oxide and oxygen in equal parts. In some cases cyclopropane has replaced this mixture during the latter part of the operation. In order to inflate one lung selectively while operating on the other, a double-lumen Carlens' tube[3] was tested; it worked satisfactorily, but is not necessary. Despite the use of controlled respiration throughout with a single-lumen tube, exposure is not obstructed.

The patient lies supine on a roll which elevates the chest (Fig. 1, *A, B, C*) and a transverse anterior incision is made as illustrated in Fig. 1, *A*. In women, the breasts nearly hide the resulting scar. In men, this cosmetic result cannot be achieved so the incision is placed above the nipples. In contrast to a posterolateral incision the muscles of the chest wall beneath this incision suffer little trauma. It is necessary only to split the pectoralis muscles in order to reach the third interspace. This interspace is then opened bilaterally before the sternum is divided transversely. A self-retaining retractor in the midline with another supplementing it on the side under attention provides good exposure (Fig. 1, *D* and *E*). Occasionally the lower sternal segment will press posteriorly against the heart as the wound is opened but this can be corrected by patiently adjusting the position of the retractor blades.

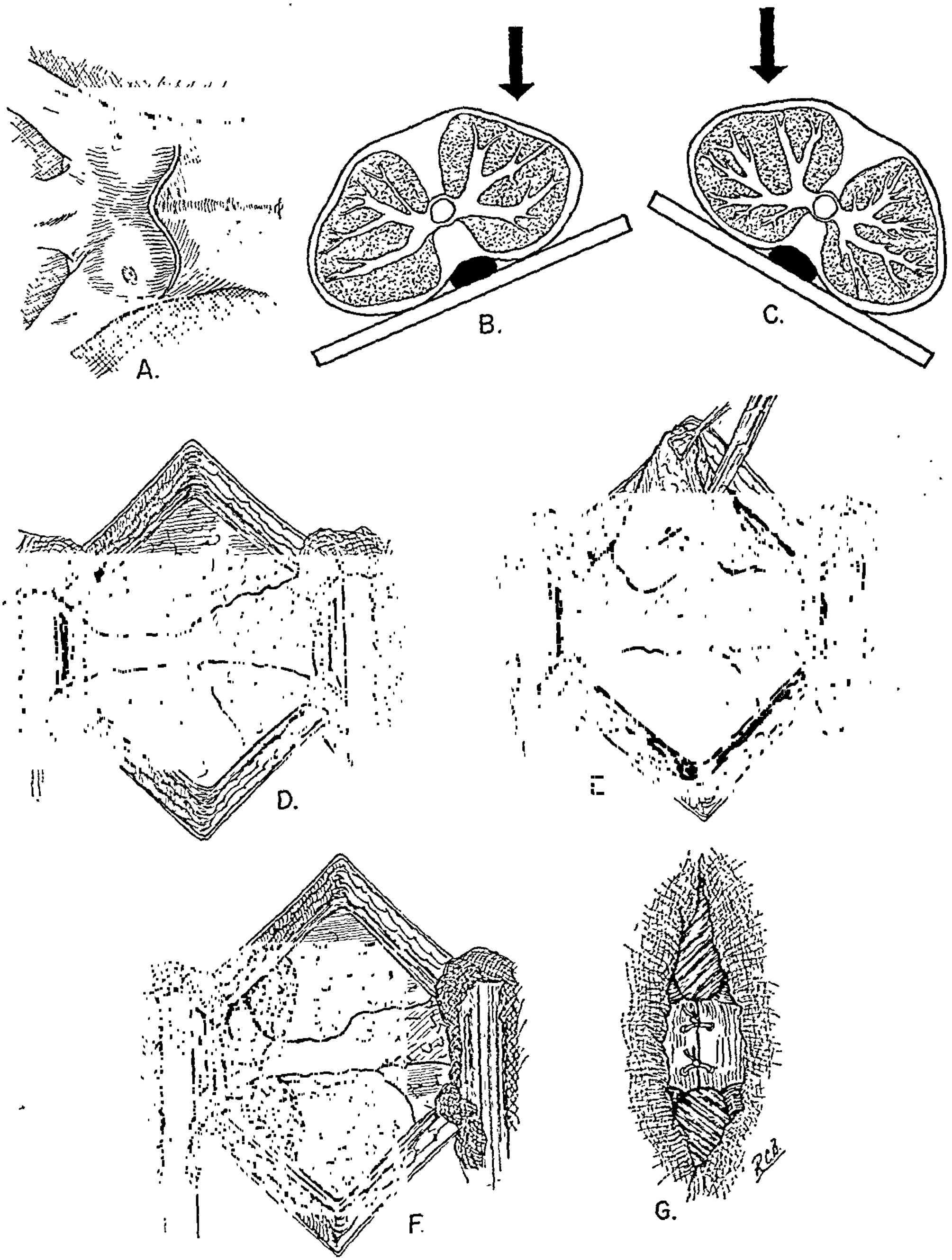

Fig. 1.—Technique of the simultaneous bilateral operation.

A, Position of the patient. The location of the incision in a woman is illustrated. Arms are usually kept at the sides.

B, *C*, Rotation of the table first to one side and then to the other in order to obtain maximum exposure. A roll of cloth is placed under the back to elevate the center of the chest.

D, Exposure obtained upon opening the wound.

E, Resection of the left apical-posterior segment is in progress. Better exposure is obtained if a second retractor is inserted and if the table is rotated to the patient's right. The raw lung surfaces are illustrated.

F, Resection has been completed on both sides.

G, Sternum approximated with heavy wire sutures.

Resection of both apices can now proceed at the same time with two operating teams but we usually have not had enough help for this. In any case, by doing one side at a time after rotating the table appropriately (Fig. 1, *B* and *C*) a wider field is exposed.

Other authors have described resection techniques in detail[4, 17, 19] so little need be added here. Approach from the front alters none of the essential steps. After cutting the adhesions, we divide the arteries to the diseased segment and then the bronchus. Following this the segment is separated from the rest of the lobe as near as possible to the intersegmental plane, mostly by blunt finger dissection starting from the root. Small veins that cross the intersegmental plane are clamped as they are encountered.

After the resections are completed (Fig. 1, *E*), two large intercostal catheters are fixed in each chest and the wound is closed. The sternum is wired with heavy stainless steel (Fig. 1, *G*) and the muscles are brought together with gut. The entire operation requires but little longer than a unilateral resection because the wound takes less time than a posterolateral wound.

POSTOPERATIVE CARE

Postoperative care demands special vigilance. The mental attitude of the insane always complicates care following any operation and a bilateral thoracotomy obviously adds special hazards. Chest catheters are stitched in place with fishline so that they can be pulled out only with great difficulty and the catheters of each side are connected to separate suction systems. When the lung fails to fill the hemithorax, an additional catheter may be inserted high, anteriorly through a trocar.

Many mental patients do not obey. They may remain passive and indifferent or they may resist treatment actively. In either case, if they do not cough forcefully, a tracheotomy should be made to permit frequent tracheal aspiration.

After recovering from surgery, the patients return to the preoperative routine. Antibiotics are continued for at least four months postoperatively and long enough, in any case, so that a total of twelve months pre- and postoperative treatment has been given. The patients are not kept in bed.

Nine months or more following surgery these patients may be discharged from isolation if they are clinically well, if their x-ray examinations are satisfactory, and if they have had at least 6 negative and no positive gastric culture examinations following discontinuance of antibiotics. Further follow-up conforms to a routine described previously.[12]

CLINICAL MATERIAL

The simultaneous bilateral operation has been done on 16 patients with no deaths. The sex distribution and age range are shown in Table I. The entire group of 187 mental patients who have had pulmonary resections for tuberculosis averaged 46.6 years in age—only a little older than the bilateral group.

TABLE I. SIMULTANEOUS BILATERAL RESECTION FOR PULMONARY TUBERCULOSIS

NUMBER OF PATIENTS	SEX		AVERAGE AGE	AGE RANGE	DEATHS
	M	F			
16	11	5	44	25 to 56	0

One patient was a convict transferred to the tuberculosis unit from prison while the others carried psychiatric diagnoses. Ten had schizophrenia and they were further classified as follows: paranoid, 6, hebephrenic, 3, and catatonic, 1. Three were feeble-minded 1 was a manic depressive, and 1 bore the diagnosis of chronic brain syndrome plus a nonspecified convulsive disorder.

All but one of the operations conformed to our plan of employing the simultaneous bilateral operations for patients who required no more than segmental excisions on each side. The commonest resection, used in 9 patients, was an apical-posterior segmental resection on the left with a resection of both the apical and posterior segments on the right. In 5 patients more lung tissue was removed while in 2, lesser resections proved adequate. Of the 5 in whom more tissue was removed, 2 had the operation just described plus wedge resections of the lingula or superior segment, 1 had the first operation plus resection of the right superior segment, and in 1, resection of the left anterior segment as well as a wedge of the superior segment was added. The fifth had a right upper lobectomy and a wedge resection of the superior segment plus resection of the left apical-posterior segment. One of the 2 patients who required less than the first operation described had an apical-posterior segmental resection on the left but only an apical segmental resection on the right. The other had an apical and posterior segmental resection on the right plus multiple wedge resections on the left.

Ten patients convalesced uneventfully while 6 suffered various significant complications. One man, while unguarded, pulled out all the chest tubes during the first postoperative night and he was, moreover, troubled by retained respiratory secretions. After new tubes were inserted and a tracheotomy was performed he recovered satisfactorily. Two other patients failed to cough effectively and atelectasis developed. Following tracheotomy both of them were relieved of their respiratory difficulty and further trouble was averted.

Finally, the last 3 patients operated upon, all men, have had a complication peculiar to the operative incision used. In each of them the sternal repair broke down. The skin remained intact in 1 and though there was some paradoxical chest motion he was helped by a tracheotomy and maintenance of a flexed posture. He had no further complications. In the second patient the skin separated after the sternum came apart and the wound opening led directly into the superior part of the left pleural cavity. The entire space became infected and in time a bronchopleural fistula appeared. He was treated with a tracheotomy, drainage, and a partial thoracoplasty; the space disappeared and the fistula has finally closed. The last patient in the series also had an anterior wound separation and infection following a sternal breakdown. To begin with, this man was a poor operative risk because he had diffuse, moderately severe pulmonary emphysema and was 52 years old. Despite this trouble he is now recovering rapidly, though the wound is not quite healed. The separated sternum was brought together by keeping him in a sitting position with the thorax flexed. Respirations were further strengthened by using an abdominal binder and by doing a tracheotomy. Fortunately, the pleural cavities were not involved in the wound breakdown.

Since the first of these operations was done only fifteen months ago, its effectiveness in eradicating the disease cannot yet be measured. Up to the present time, however, none of the patients have suffered reactivation of their disease. Four have been discharged from the tuberculosis unit, 1 is awaiting discharge, 5 receive antibiotics no longer and they are undergoing a series of gastric washings for culture examination or await reports on these examinations, and the last 6 patients are still being treated with antibiotics.

DISCUSSION

In our complete series the incidence of bilateral resections, either staged or simultaneous, has been high. Among a total of 187 patients with resections, 26 have had staged bilateral resections and 16 (the present group) have had a simultaneous bilateral operation. Thus 22 per cent of the entire group have had bilateral resections and a few more, still in the unilateral group, may be candidates for a contralateral resection. We have employed resectional surgery freely in this special type of patient—State Mental Hospital inmates with tuberculosis—for reasons discussed earlier in the paper. As a consequence we have apparently found a greater need for bilateral resections than others.[4, 6-10, 20] It is not yet established that residual caseous nodules of tuberculosis should all be resected but at least for our special group of patients we have assumed that this is the best course. If it is, many patients will need bilateral resections and so it is reassuring to know that they can be done with a low risk. Though the fatality rate in the entire group of 187 patients who underwent resection for tuberculosis was 8.6 per cent there were no deaths among the 42 patients who had staged or simultaneous bilateral resections. It must be pointed out, however, that two deaths in the unilateral group were in patients who had anterior incisions similar to the incision used for the bilateral operations. Both succumbed on the thirteenth postoperative day, one of a pulmonary embolism, while the other had bronchopneumonia and atelectasis.

Sternal separation as a postoperative complication was unexpected. A similar sternal repair did not break down in any of the 27 patients whose atrial septal defects were repaired through this type of incision.[13] For the heart operations the sternum was usually divided at the fourth rather than the third interspace and there may be less strain at this lower level, but this seems unlikely. There is another possible explanation. Insane patients often react minimally to painful stimuli and they may, therefore, fail to protect an operative wound as carefully as sane patients do. While agitated, insane patients move strenuously, treating their operative wounds with apparent indifference. Whatever the cause, sternal separation can probably be prevented, even among mentally agitated men, if a stronger sternal repair can be devised.

SUMMARY

1. A simultaneous bilateral resection has been used for pulmonary tuberculosis in 16 inmates of a state mental hospital. There were no deaths.

2. The lungs were exposed through a single anterior, transverse incision with transection of the sternum at the third interspace. Except for the incision, technical details of the resections are those of the conventional operation.

3. Sternal separation postoperatively complicated the recovery of 3 patients.

4. Tuberculosis has not reactivated as yet in any of the patients but the first patient was operated upon only fifteen months ago.

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DISCUSSION

DR RICHARD H MEADE, Grand Rapids, Mich —I rise not to discuss the merits of this paper, which I think is an excellent presentation, but I want to be the first to say that I think this is the best presentation we have had Here is a man who has talked to you, who has not read a paper I hope the time will come when this organization can have all papers presented in this manner

DR G N J SOMMER, Jr, Trenton, N J—I think it is a very wonderful thing to have this subject—the treatment of tuberculosis in the mentally ill—discussed before this Association We all know it is a serious problem that is very much neglected I believe it is a problem that can be solved The solution will come through a treatment program in every mental hospital and by a very careful program of detection I am rather surprised to read that, in New York State, the mental institutions have had four or five surveys in the last seven or eight years I believe it is important that a survey of all the patients and employees be done twice a year in each hospital In our own experience in the State Hospital at Trenton, we have, I believe, cut down our incidence of new cases developing among our own patients tremendously by this program, and by proper isolation of the patients A great many of our patients with severe tuberculosis are now admitted from the outside rather than developing tuberculosis within the institution

To get down to Dr Lewis' paper, I am much impressed by the total number of patients operated upon I am not surprised in this particular operation of bilateral resection that the three most serious complications occurred in the last 3 patients That has been my experience About the time I thought that I could get as good results generally with psychotic patients as with nonpsychotic patients, I ran into trouble Quite frankly, I am not in sympathy with the bilateral operation, because I have an idea that if he continues he is going to have a greater percentage of serious complications than he thought he would have in the beginning The last 3 patients were those who had sternal separations

In my own experience these are difficult patients to operate upon That does not deter me and I hope it does not deter any others, but one is certainly going to have more trouble I have had enough trouble with unilateral operations without going into bilateral operations at one sitting As Dr Lewis pointed out, the average age of these patients is higher than in nonpsychotic patients upon whom we all operate I have the feeling that we could go back in some of these patients and do thoracoplasties, because in the older group we find more serious emphysema This is my experience, and in talking to some of the members of this Association I understand they have had similar troubles, it leads to more difficulty with air leaks and eventually with bronchial fistulas from the unexpanded lung For those patients, I have an idea that thoracoplasty offers a safer method of operation

Several years ago, with my colleagues in two hospitals, we reported a number of cases of thoracoplasties with good early results These results have stood up very well, and I believe that this is an indication that in a number of older patients we should go back to this procedure, once so popular

DR RICHARD H OVERHOLT, Boston, Mass —Since one is often confronted with the problem of bilateral surgical disease, it is fortunate that we have heard this presenta tion today. It may be unfortunate that there are not psychiatrists present so that they might hear of a new type of shock therapy. There is no question but that certain patients with bilateral pulmonary disease may be saved time, money, and considerable discomfort if a bilateral procedure can be accomplished at one sitting. Dr. Lewis and his group have been confronted with a captive group of patients. Our experience has been with patients who voluntarily elect to come. This has forced us to be deeply concerned with morbidity, mortality, complications, total discomfort, and expense.

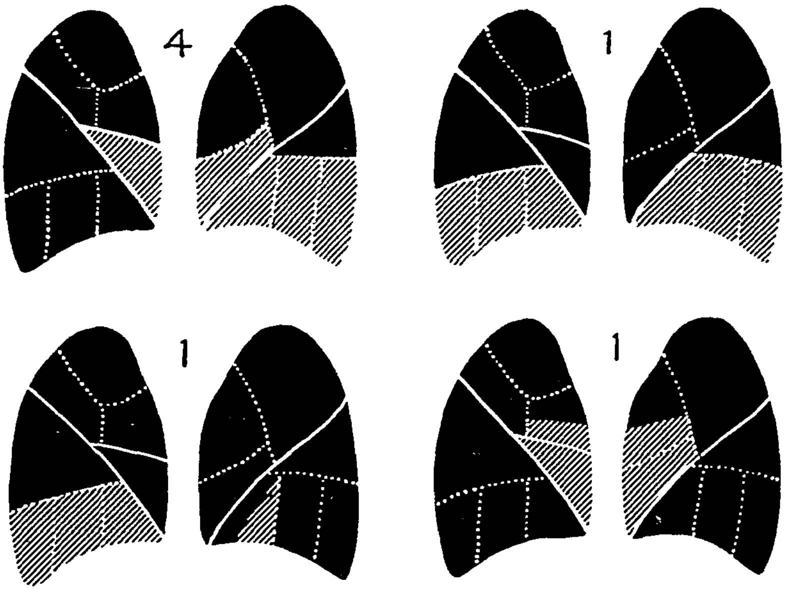

Fig. 1.—Simultaneous bilateral resection—bronchiectasis.

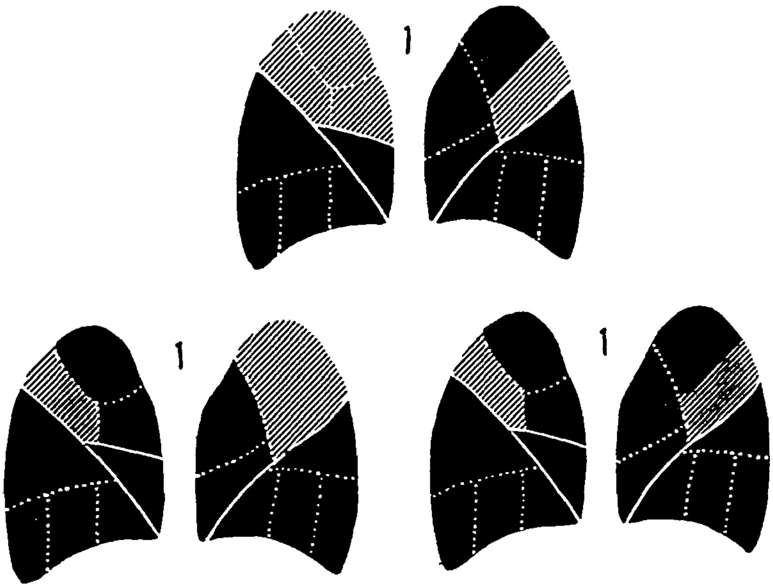

Fig. 2.—Simultaneous bilateral resection—tuberculosis.

SIMULTANEOUS BILATERAL RESECTION

Total 10

DATE	DIAGNOSIS	NUMBER
1952	Bronchiectasis	3
	Tuberculosis tumor	1
1953	Bronchiectasis	1
	Tuberculosis	2
1954	Bronchiectasis	2
1955	Bronchiectasis	1

Our first operation was performed in January, 1952. This was for bronchiectasis involving both right and left anterior segments, the middle lobe and the lingula.

The extent of the resection in the 7 bronchiectasis cases is shown. Four cases had an identical pattern. In this group, we elected to do the right side first and then, if everything went well, we proceeded immediately with the second, or left side.

The extent of resection in the 3 cases treated by simultaneous resection for tuberculosis is shown. The patient in whom only the posterior segments required removal was a young patient with diabetes which was difficult to control. She was four months pregnant. There was active cavitary disease. The direct approach appeared to be the surest, safest, and quickest way to control the tuberculous complicating factor.

SIMULTANEOUS BILATERAL RESECTION

Total 10

Mortality		0
Morbidity	P. O. hospital stay	
Average	15 days	
Longest	26 days*	
Shortest	10 days	

There was no mortality and the hospital stay was not significantly longer than for patients undergoing unilateral surgery.

Dr. Lewis has advised an anterior approach with a division of the sternum. There are reasons why I would not use this method. In the first place, both cavities are opened in the beginning before it can be decided whether or not it is wise to proceed with both resections simultaneously. Second, it is safer to have one side sealed before opening the contralateral cavity. Third, the division of the sternum is accompanied by more interference with costal-cage action and more pain postoperatively than is found with two independent posterolateral wounds.

DR. JOHN PETTER, Rutland Heights, Mass.—The possible advantages of simultaneous resection, particularly in mental patients, are recognized. We believe, however, that the added risk of simultaneous bilateral complications makes staging of the procedures a safer program for the average patient. Our reasons for adopting a more conservative program are as follows:

1. Studies in our laboratory and in others have shown that although the loss of function following resection of up to five segments is usually very small, this loss may be significantly greater if pleural or bronchial complications develop.
2. These complications, though infrequent in well selected cases, remain unpredictable in any individual case.
3. In the event of a significant functional loss following the first resection, interval pulmonary function studies may prove helpful in deciding whether or not the contralateral resection may be safely undertaken. If not, the original plan may be changed, either in selection of a procedure with lower risk of added function loss, or no further surgery.
4. The loss of time by staging the second procedure rarely exceeds two months.
5. Further resolution of the remaining disease after the first resection may preclude the need for a second operation.

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*Only 1 complication—empyema.

(slide) I would like to present briefly our series of 32 patients who have had staged bilateral surgery at Rutland Heights Veterans Administration Hospital. Twelve patients had bilateral resection, 10 had bilateral thoracoplasty, 9 had resection on one side, thoracoplasty on the other, and 1 patient had decortication followed later by contralateral thoracoplasty. Complete pulmonary function studies were performed on all patients prior to operation on the contralateral side.

(slide) There were no operative deaths and no late deaths due to tuberculosis. Two patients, both with bilateral thoracoplasties, have died at home of nontuberculous causes three years after the second operation. All patients have been followed to date. There are no pulmonary cripples. Twenty-one are at home with negative sputum, one positive. Eight patients recently operated upon are still hospitalized, six negative, two positive.

In summary, we agree that bilateral surgery should be performed when necessary, but we believe that even in carefully selected cases, staging of the procedures with interval pulmonary function studies is a safer and more widely applicable policy.

DR. DENTON A. COOLEY, Houston, Texas.—I congratulate Dr. Lewis on a most interesting paper which poses many fascinating and challenging problems. We have used bilateral simultaneous thoracotomy in patients with recurrent spontaneous pneumothorax occurring bilaterally. It is well known that anterior intercostal incisions provide adequate exposure for pulmonary resections without transecting the sternum. Failure of sternal healing could be a serious complication and one which in this case is avoidable without interfering appreciably with the surgical exposure by employing separate incisions.

DR. LAURENCE H. RUBENSTEIN, Chicago, Ill.—I wish to congratulate the authors on their bold attack on tuberculosis control in mental institutions. In the State of Illinois Mental Hospitals (Kankakee State Hospital), we also feel strongly about surgical treatment for tuberculosis in the mental patient and have embarked on a surgical program. While we have not done simultaneous bilateral resections we have encountered every type of surgical problem and have been pleased with the success of such a program. In more than 150 cases we have had no deaths. In order to eradicate and control tuberculosis in mental institutions we feel an energetic surgical program must be instituted.

DR. LEWIS (Closing).—I should like to thank the discussants for their kind and generally tolerant remarks. I expected a good deal of trouble, actually. I appreciate Dr. Cooley's suggestion about using the anterior approach for other types of lung surgery. We have considered it but have not had suitable cases. I would like to point out that that incision would probably not be as suitable for lower lung resections, the type Dr. Overholt has done through two incisions, because the heart, especially on the left side, seems to obstruct a good view of the lower part of the lung root.

I cannot reply to all the suggestions or answer all the implied criticisms. We have not had the facilities for doing lung function studies on these cases, but I do feel that the anterior incision actually, if the sternum does not separate, is a much easier incision to bear than a one-sided posterolateral incision.

BLOOD VOLUME STUDIES IN PULMONARY TUBERCULOSIS

An Analysis of 100 Cases

John H. Kehne, M.D. (by invitation), Felix A. Hughes, M.D., and
Frank S. Schlenker, Ph.D. (by invitation)
Memphis, Tenn.

THE importance of blood volume deficiencies in the chronically ill patient has been stressed in previously published reports[1-5] but limited observations have been made in patients who have chronic pulmonary tuberculosis. Despite general knowledge of the problem, designated by Clark and associates[1] as chronic shock, patients with chronic pulmonary tuberculosis continue to be transferred to the Thoracic Surgical Section of the Veterans Administration Medical Teaching Group Hospital (Kennedy) who, despite long periods of hospitalization and treatment, present clinical manifestations of blood volume deficiency. These symptoms commonly occur in patients with pulmonary tuberculosis, but are usually interpreted as manifestations of the underlying pulmonary disease. A survey study of blood volume was undertaken to determine the frequency and magnitude of the existing deficiencies. The present report is an evaluation of the blood volume studies in 100 unselected patients with pulmonary tuberculosis who were considered candidates for thoracic surgical procedures

METHOD

Plasma volume was determined by the Evans blue dye method as described by Gregersen[7] and modified by Gibson and Evelyn[8] for the photoelectric colorimeter.

Hematocrit values were determined by the Wintrobe method and utilized in calculating the total blood volume. The normal expected values for blood and plasma volumes were found by multiplying the normal average pre-illness weight expressed in kilograms by 85 ml. per kilogram and 45 ml. per kilogram for blood and plasma volumes respectively.[8] In the group of patients in whom obesity was a factor, prior to illness, the ideal weight based upon the standard weight tables for sex, age, and height were used.[1] Total hemoglobin mass was determined by the formula

$$\frac{\text{Total blood volume} \times \text{Hgb}/100 \text{ c.c.}}{100}.$$

Deficiencies for blood volume, plasma volume, and total hemoglobin mass were determined by subtracting the calculated values from the expected values based upon the normal or ideal weight.

From the Thoracic Surgical and Laboratory Services, Veterans Administration Medical Teaching Group Hospital, Memphis 15, Tenn

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 24 to 26, 1955.

CASE ANALYSIS

The patients included in the surveyed groups varied in age from 21 to 64 years. All patients had an established diagnosis of pulmonary tuberculosis varying from one to twelve years and had periods of continuous hospitalization varying from nine months to four years prior to the blood volume studies. These patients were transferred to the Thoracic Surgical Section of Kennedy Hospital from pulmonary disease sections of five hospitals in the South. Selection was made from consecutive admissions of patients who were considered to be candidates for either resectional or collapse therapy as an adjunct to their course of tuberculosis treatment.

RESULTS

In Table I are presented the mean values for blood and plasma volume and total hemoglobin mass for a group of 25 normal male adults, and 100 patients with chronic pulmonary tuberculosis considered to be candidates for thoracic surgical therapy. Blood and plasma volumes are expressed in terms of cubic centimeters per square meter of body surface and total hemoglobin mass in grams per square meter of body surface. The 100 tuberculous patients were subdivided into two groups: (a) 38 patients who were found to have no significant deficit in blood or plasma volume and hemoglobin mass, and (b) 62 patients who had significant deficits. Deficiencies of less than 500 c.c. of whole blood were considered to be within the limits of laboratory error and were included with the nondeficient group.

TABLE I. BLOOD AND PLASMA VOLUME DETERMINATIONS

GROUP	NO.	MEAN BLOOD VOLUME C.C./SQ.M.	MEAN PLASMA VOLUME C.C./SQ.M.	MEAN HEMOGLOBIN MASS GM./SQ.M.
I. Normal adult	25	2971	1690	447
II. Tuberculosis	100			
normal—Group A	38	3064	1668	466
deficient—Group B	62	2710	1532	379
Group difference (A − B)		354	136	87

Blood Volume.—The mean blood volume for the normal-control group was found to be 2,971 c.c. per square meter body surface and 3,064 c.c. per square meter body surface for the tuberculosis group (A) which was normal in volume (Table I). The difference is not statistically significant. The mean blood volume for the deficient tuberculosis group (B) was 2,710 c.c. per square meter body surface, a group difference (A minus B) of 354 c.c. per square meter body surface. Although numerically small, this value was an average deficiency of 12 per cent in total blood volume.

Plasma Volume.—Comparable mean plasma volumes were found for the normal-control group and the nondeficient tuberculosis group (A), the values being 1,690 c.c. per square meter body surface and 1,668 c.c. per square meter body surface, respectively (Table I). In the deficient group (B), the mean plasma volume was found to be 1,532 c.c. per square meter body surface, representing an average group difference (A minus B) of 136 c.c. per square meter body surface or 8 per cent average plasma volume deficiency (Table I).

The mean values for both blood and plasma volume have been presented graphically in Fig. 1. It is to be noted that the mean values for the normal-control group and nondeficient tuberculosis group are comparable. In the deficient group, however, it can be seen that the decrease in blood volume has exceeded the fall in plasma volume and represents a relative increase in plasma volume of 4 per cent. In no case was the laboratory value for plasma volume found to exceed the calculated plasma volume, and attempts to correlate either blood or plasma volume with disease activity or nutritional status were unsuccessful.

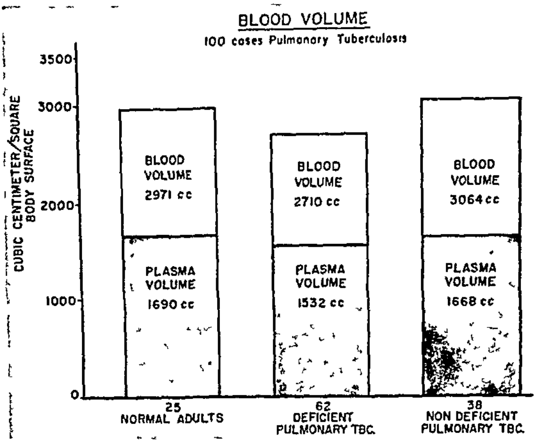

Fig. 1.

Total Hemoglobin Mass.—Comparison of the mean hemoglobin mass for the normal-control group (447 Gm. per square meter body surface) and the nondeficient tuberculosis group (466 Gm per square meter body surface) reveals no significant difference (Table I). The average deficiency in total hemoglobin mass for the deficient tuberculosis group was 87 Gm. per square meter body surface or an average deficiency of 19 per cent.

The values obtained for total hemoglobin mass have been presented graphically in Fig. 2 for the 100 cases of pulmonary tuberculosis. The deficient group of 62 cases was further subdivided into Group A, deficient by calculation but without excessive weight loss and only moderate pulmonary involvement, and Group B with weight losses in excess of 20 per cent body weight or with more extensive pulmonary involvement than was seen in Group A. The mean hemoglobin mass for the entire group of 62 cases found to be deficient was 379 Gm. per square meter body surface (Fig. 2). Thirty-one cases could be classified in Group A with an average total hemoglobin mass of 378 Gm. per square meter body surface and 31 cases in Group B with an average hemoglobin mass of 381 Gm. per square meter body surface. Although the major individual case deficiencies in total hemoglobin mass were found in Group B, this is not apparent from the average values, indicating either a considerable group variation or a poor correlation with surface area.

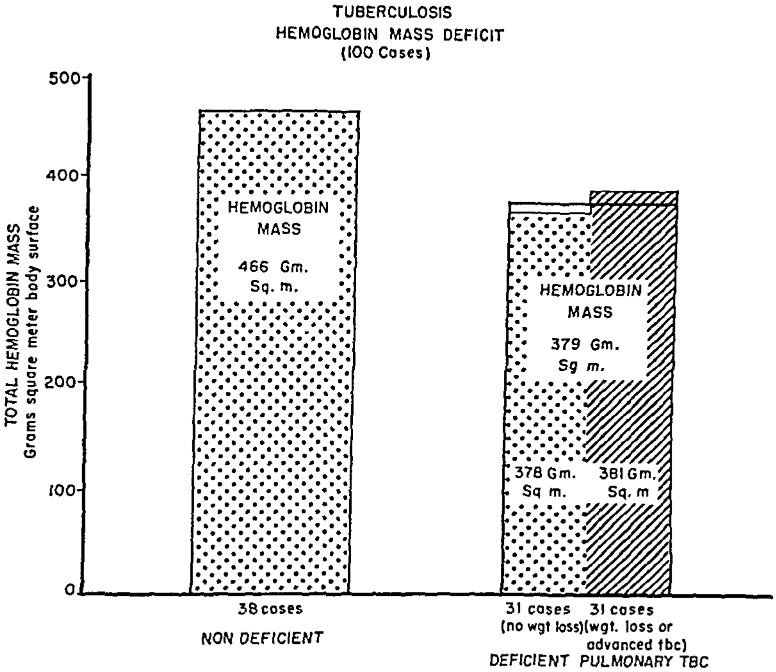

Fig. 2.

Inasmuch as the major deficiency between the normal-control group and the deficient tuberculosis group was in total hemoglobin mass with an average deficiency of 19 per cent, this value was selected for further analysis. In view of the average decrease in blood volume of 12 per cent in the deficient tuberculous group, it is apparent that total hemoglobin mass replacement would be limited by the deficiency in total blood volume. Therefore, replacement therapy should consist of both whole citrated blood and washed red blood cells suspended in isotonic saline solution.

Fig. 3 presents the hemoglobin mass requirements for the 100 tuberculous patients included in this study. The deficiency is expressed in the hemoglobin equivalent of 500 c.c. of blood (15 Gm. hemoglobin equivalent to 100 c.c. whole blood). Sixteen patients required the hemoglobin equivalent of 500 c.c. of whole blood; 23 patients required 1,000 c.c.; 15 required 1,500 c.c.; 5 patients 2,000 c.c.; and 3 required 2,500 c.c. In summation, 62 per cent of the 100 patients required the hemoglobin equivalent of 500 c.c. or more of blood; 46 per cent required 1,000 c.c. or more; 23 per cent required 1,500 c.c. or more; and 8 per cent required 2,000 c.c. or more.

In each of the 38 cases included in the nondeficient group (A) the hemogram was normal. In the 62 cases with blood volume deficiencies (Group B), normal values for red-cell count, hemoglobin, and hematocrit were reported in 50 per cent, and the remaining 50 per cent were below the expected normal values. The reported laboratory values in general failed to reflect the degree of deficiency either in blood volume or hemoglobin mass. The variation in blood volume and plasma volume seen in Fig. 1 has been described elsewhere[1, 6, 9] and explains why routine laboratory values misrepresent the true hematologic status.

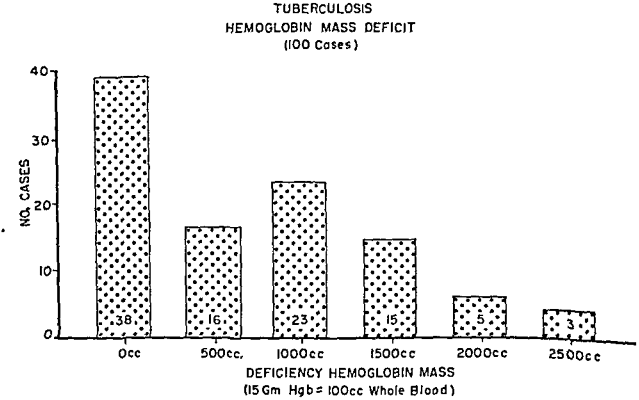

Fig. 3.

It has been reported[1] that a clinical history of weight loss has consistently indicated a reduction in blood volume and total circulating hemoglobin mass. The grouping of the 38 cases of tuberculosis with normal blood volume and 62 cases deficient in blood volume with reference to weight loss is shown in Table II. There were 12 cases (32 per cent) in which the recorded weight loss was 10 per cent or more in the nondeficient group and 23 cases (37 per cent) in which the weight losses were 10 per cent or more in the deficient group.

Table II. Pulmonary Tuberculosis (100 Cases)

WEIGHT LOSS	0%	10%	20%	30%	40%
Normal blood volume	26	7	5	0	0
Deficient blood volume	39	9	12	1	1

However, the major portion of these was in excess of 20 per cent total weight loss. It is apparent that weight loss fails to identify the patient with a blood volume deficiency, but when a major weight loss is present a deficiency must be suspected.

CASE REPORTS

Case 1.—A 27 year old white man had had a diagnosis of pulmonary tuberculosis since October, 1952. His height was 68 inches; average normal weight, 125 pounds. *Initial therapy* consisted of bed rest, streptomycin and isoniazid in combination with para aminosalicylic acid. The sputum was persistently positive for acid-fast bacilli. *On Feb. 3, 1953* right decortication and upper lobectomy were performed. The postoperative course was complicated by a persistent bronchopleural fistula which was controlled by a right 5-rib thoracoplasty on Feb. 20, 1953; modified Schede thoracoplasty on March 13, 1953, and final revision thoracoplasty on April 27, 1953. During this period of treatment, a weight loss of 19 pounds was recorded. At the time of transfer to another hospital for continued treatment, hemoglobin was reported as 11.8 Gm./100 c.c. During the ensuing two months, he failed to gain weight and hemoglobin values of 11 to 12 Gm./100 c.c. were recorded. No effort was made to correct his deficiency. He was returned to Kennedy Hospital for treatment of right costal-arch chondritis and draining sinus on July 20, 1953. The sputum had remained negative for acid-fast bacilli after the lobectomy.

Examination revealed a pale, apathetic, white man appearing chronically ill. The superficial veins of the extremities were collapsed. It was noted that he spent much of his time in bed and his appetite was poor.

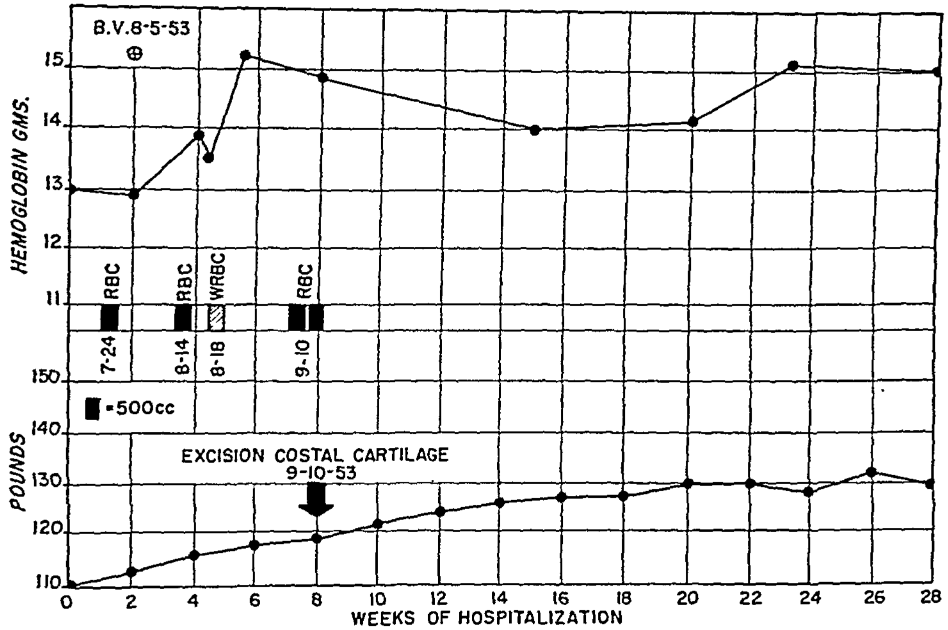

Fig. 4.

Laboratory Data (July 21, 1953).—Red-cell count, 4.9 million; hemoglobin, 13 Gm.; hematocrit, 43 mm. Because of these findings, an initial transfusion of 500 c.c. of whole blood was given after which blood volume studies were done using the T-1824 or Evans blue dye method. A residual deficiency of 573 c.c. or 16 per cent in blood volume was found and a deficiency of 192 Gm. of hemoglobin or 24 per cent of the expected hemoglobin mass. He was given 500 c.c. of whole blood and 500 c.c. of washed red blood cells suspended in saline solution. The weight-laboratory correlation is recorded in Fig. 4. There was an immediate return of appetite, requiring an increase in caloric intake to 5,000 calories, and a progressive weight gain became apparent. He became jovial, ambitious, and overly active, making enforcement of the rest requirement difficult. Surgery was performed for the correction of the osteochondritis and chronic draining sinus; and at the time of discharge on Jan. 15, 1954, he had regained 21 pounds in weight.

Comment.—This patient had developed an accumulative blood volume deficiency following a series of operations for removal of the right upper lobe of the lung and control of a complicating bronchopleural fistula. Satisfactory restoration of blood volume had never been accomplished. Despite consistently negative sputum following the initial procedure, the manifestations of a blood volume deficiency persisted, i.e., pallor, superficial venous collapse, apathy, anorexia, lack of enthusiasm and energy, easy fatigability, and inability to regain a major weight loss. It is possible that with the passage of time the deficiency would have been corrected, but it is apparent from our observations that the patient's clinical course was rapidly reversed by volumetric blood volume replacement.

CASE 2.—A 28-year-old Negro man was admitted on Sept. 6, 1951, following six months of bed rest for diagnosed pulmonary tuberculosis. X-ray examination of the chest revealed far-advanced involvement of both lungs. The sputum was positive for acid-fast bacilli. Laboratory work at the time of admission: red-cell count, 2.3 million; hemoglobin, 6.9 Gm. The patient received streptomycin, 1 Gm. daily; PAS, 12 Gm. daily; and Chloromycetin, 1 Gm. daily. He was given 1,000 c.c. of citrated blood. Thereafter, he developed resistance to streptomycin and PAS and was given subsequent courses of aldinamide, INH, and viomycin. Resistance developed to all drugs with the exception of viomycin. Bronchoscopy was performed which revealed a profuse mucopurulent bronchial secretion and acute inflammatory changes involving the left main bronchus. The patient remained seriously ill and developed a psychosis manifested by a fetal position in bed accompanied by speech and thought impairment. His general condition thereafter slowly improved. The sputum remained positive for acid-fast bacilli by smear and culture. During the period of acute illness, he developed flexion contractures of both knees which were partially corrected by active and passive exercises and the use of plaster splints. He had been completely bedfast for approximately one year prior to the time of transfer to the Thoracic Surgical Section on Jan. 8, 1954. At that time, examination revealed a mentally dull, apathetic, Negro man with evidence of marked muscular wasting. There were pallor of the conjunctiva and mucous membranes and evidence of peripheral superficial venous collapse. *It was estimated that his weight was approximately 100 pounds in contrast* to his normal weight of 160 pounds. X-ray examination of the chest revealed far-advanced bilateral tuberculosis with extensive cavitation of the left upper lobe.

Laboratory Data (Jan. 8, 1954).—Red-cell count, 4.22 million; hemoglobin, 11.4 Gm.; hematocrit, 37 mm.; sedimentation rate, 34 mm. per hour corrected. A blood volume determination was performed on Feb. 10, 1954, utilizing the T-1824 method and the following deficiencies calculated: total blood volume, 993 c.c. or 16 per cent of normal; plasma volume, 210 c.c. or 6 per cent of normal; total hemoglobin mass, 269 Gm. or 28 per cent of normal. It was estimated that blood replacement requirements would be 500 ml. of whole blood plus 1,500 ml. of washed red blood cells suspended in normal saline solution.

Transfusions of 500 c.c. of citrated blood were given on Feb. 12, 1954, and on Feb. 15, 1954, followed by a total of 1,500 c.c. of washed red blood cells.

After the final transfusion, the following laboratory reports were recorded: red-cell count, 5.9 million; hemoglobin, 15.6 Gm.; hematocrit, 49 mm. The patient became mentally alert, physically stronger, and he required a 5,000 calorie diet as his appetite improved remarkably. He was able to sit in a wheelchair daily for increasing periods. A progressive weight gain was noted for the first time. It was possible to perform a left anterior-stage thoracoplasty on Feb. 18, 1954; first-stage posterior thoracoplasty on March 15, 1954; second-stage posterior thoracoplasty on April 20, 1954; and third-stage posterior thoracoplasty on May 18, 1954. Adequate blood transfusion replacements were given during each operative procedure and all procedures were tolerated well. The sputum remained positive and the patient was returned to the Medical Tuberculosis Section on June 3, 1954. At that time, the red-cell count was 5.02 million; hemoglobin, 13.8 Gm.; hematocrit, 44 mm. During the subsequent period of observation, the patient has continued to have an excellent appetite and his mental outlook has been markedly improved.

He has regained approximately 40 pounds in weight. Sputum examinations have remained positive for acid-fast bacilli by concentration and culture. Because of far-advanced bilateral pulmonary tuberculosis, he was not considered a candidate for further surgical therapy.

Comment.—This patient had shown a poor response to therapy until the time of blood volume replacement. He had lost an excessive amount of weight and had been bedfast for approximately one year. Despite a calculated total blood volume deficiency of 993 c.c. and a total hemoglobin mass deficiency of 269 Gm., it was found that 1,000 c.c. of citrated blood and 1,500 c.c. of washed red blood cells were required. Following the total replacement of approximately 375 Gm. of hemoglobin, the laboratory values returned to the normal range and the patient's clinical course was reversed despite the presence of far-advanced active pulmonary tuberculosis.

DISCUSSION

Analysis of the blood volume, plasma volume, and hemoglobin mass deficiencies in this group of 100 cases of pulmonary tuberculosis revealed that 62 per cent of the total group had replaceable hemoglobin deficiencies equivalent to 500 c.c. or more of blood. Sixteen per cent of the total group required one transfusion of 500 c.c. (Fig. 2). In this group, there were no clinical manifestations of the deficiency and this would be of importance only when computing blood replacement requirements at the operating table. Forty-six per cent of the group required the hemoglobin equivalent of 1,000 c.c. or more of blood. These are major blood volume deficiencies and the clinical manifestations of the deficiency are usually present. This figure becomes more significant when interpreted in terms of expected total hemoglobin mass when a 1,000 c.c. deficiency could represent a deficiency of 20 to 30 per cent of the normal value in the individual case. It was surprising to find that 23 per cent of the total group required 1,500 c.c. or more of blood and 8 per cent required 2,000 c.c. or more. It is apparent that despite continuous hospitalization and treatment for pulmonary tuberculosis, the clinical evidence of these deficiencies was mistaken for manifestations of the pulmonary tuberculosis and that the inaccuracy of the red-cell count, hemoglobin, and hematocrit had not been considered. One can only speculate concerning the duration of these deficiencies in blood volume and how the patient's clinical course would have been altered if volumetric blood replacement had been accomplished earlier in the course of the illness. It appears from the frequency of total blood volume deficiencies that this important phase of supportive treatment for a chronic illness had been neglected.

Weight loss alone failed to isolate accurately those cases with a major blood volume deficiency although it was noted that a weight loss of 20 per cent or more of body weight usually reflected a major deficiency. Examination of Tables I and II indicates that 5 patients of the group (A), 38 patients with normal calculated blood volumes, had weight losses of 20 per cent or more which makes it impossible to formulate any absolute rule relative to the weight loss–blood volume relationship.

It appears then that the best method of detecting a major blood volume deficiency is by clinical observation of the patient. The clinical picture is constant and is characterized by anorexia, lack of energy, easy fatigability, lassitude, inability to regain weight losses or maintain a constant weight, and pallor of the facies and conjunctivae. A valuable sign that we have seen with regularity is a collapse of the peripheral superficial veins. Despite the use of a tourniquet, the veins of the arm fail to distend and venepuncture is difficult. These signs and symptoms have been considered to be a manifestation of the chronic pulmonary disease. This is easily disproved because this group of signs and symptoms can be completely eliminated in the majority of cases. Total blood volume and hemoglobin mass replacement is usually followed by a return of appetite which requires an increase in caloric intake, renewed energy, and enthusiasm for life, and a reversal of the weight curve. The listless patient who spends much of his time resting in bed has been observed to become a disciplinary problem because of the additional energy which makes it difficult for him to conform to the usual rest requirements.

A quantitative method for determination of blood volume deficiency must be available. The Evans blue dye method (T-1824) was selected for the basic plasma volume determinations for the following reasons. The method is readily adapted to the average laboratory and gives reproducible results in serial determinations. Reports in the literature comparing the results of T-1824 plasma volume determinations and the I^{131} labeled human serum albumin method indicate that the results obtained by the two methods were not statistically different.[11] The reports of Storaasli and Aust and their coworkers[12, 13] suggest that the plasma volume value obtained by the I^{131} method is lower than by the Evans blue dye method. Blood studies utilizing radioactive Cr^{51} tagged red blood cells are said to yield a blood volume value 16.3 per cent lower than those obtained by the T-1824 method.[14] Since chronic pulmonary tuberculosis presents the problem of a diminished pulmonary reserve and poor tolerance of a vascular overload, the error introduced by the relatively higher value obtained by the T-1824 method and used in the calculation of the deficiency would result in an error which would tend toward undertransfusion rather than vascular overload.

Despite arguments relative to the method utilized to determine plasma and blood volume, the major problem today in the calculation of the individual deficiency is expressed by the question, "What represents this patient's normal expected blood volume?" Rowntree and Brown[15] and Gibson and Evans[16] concluded that the blood volume had a more constant relationship to body surface than to weight. Accumulated data presented by Gregersen[7] and Clark and associates[1] indicate that a close relationship exists between blood volume and body weight and that normal values for blood volume may be calculated safely from observed weight. An analysis of thirty-eight normal blood volume determinations reported here revealed an average blood volume of 81 c.c., plus or minus 9 c.c. per kilogram of body weight with a range of 54 c.c. to 98 c.c. per kilogram and an average plasma volume of 44 c.c., plus or minus 7 c.c. per kilogram of body weight with a range of 38 c.c. to 55 c.c. per kilogram of body weight. Although the average values of 81 c.c. per kilogram for blood volume and 44 c.c. per kilogram plasma volume agree with average values reported by Gregersen[7] of 85 c.c. and 45 c.c. per kilogram, respectively, and those reported by Clark[1] of 82 c.c. and 45.5 c.c. per kilogram, respectively, it is apparent from the variation of one standard deviation that the correlation with weight is poor. If the values of 85 c.c. per kilogram of body weight for blood volume, and 45 c.c. per kilogram for plasma volume are accepted, it must be realized that the calculated blood and plasma volumes must represent only an approximation of the actual value. This must be considered in volumetric blood replacement and careful control of replacement utilized in order to prevent circulatory overload. The calculation of the normal expected blood volume based upon the normal weight or ideal weight for height, sex, and age represents the greatest source of error in the calculation of blood volume deficiencies.

When the actual deficiency has been calculated and replacement requirements for whole blood and washed red blood cells have been established, a replacement plan must be utilized. It has been our policy to replace one half of the calculated deficiency without hesitation. Transfusions thereafter are given at weekly intervals at the rate of 100 c.c. per hour with careful check of the patient's blood pressure and pulse. In the interval between transfusions, red-cell counts, hemoglobin and hematocrit determinations are obtained. While these values remain within the normal expected limits, blood replacement is continued, but with the first indication of elevated values, replacement therapy is assumed to be completed. Using this system we have had no evidence of circulatory overload.

The final question to be answered then is, "Does the clinical benefit derived from total blood volume replacement offset the inherent dangers of intravenous blood transfusion today?" During the past seven years, transfusions of citrated blood have been given at this hospital with an average reaction rate of 1.65 per cent. These were classified as minor reactions characterized by chills, fever, and urticaria. There was no record of a major hemolytic reaction secondary to the administration of incompatible blood through either laboratory or handling errors. Thompson and Sutliff[17] reported 32 cases of viral hepatitis following blood transfusion from a total of 13,567 transfusions of 500 c.c. citrated blood administered at Kennedy Hospital between 1947 and 1951. Twenty of this group had also received irradiated or nonirradiated plasma. The ratios of viral hepatitis cases to units of infusion reported by those authors were irradiated plasma 1:258, nonirradiated plasma 1:116, and blood 1:424. During the past three years, there have been 2 cases of viral hepatitis following blood transfusion on our service with full recovery in each case. No major hemolytic reactions have occurred. It is felt that the clinical benefits of volumetric blood replacement far exceed the inherent dangers of transfusion.

Numerous authors[1-5] have presented the problem of diminished circulating blood volume in the chronically ill patient and the surgical significance of reduced blood volume has been stressed by Clark and co-workers.[1] This has been described as chronic shock. It has been demonstrated that restoration of blood volume must precede significant recovery in weight. Holman and associates[4] have demonstrated that when deficiencies of both hemoglobin and tissue protein exist the restoration of protein deficits can be augmented by the correction of the anemia. We have seen this principle demonstrated following blood volume restoration in our patients. In view of the surgical significance of major blood volume deficiency and its effect upon the normal reparative processes in chronic disease, the importance of replacement of blood volume deficits cannot be too strongly emphasized.

SUMMARY

Significant replaceable deficiencies in blood volume, plasma volume and hemoglobin mass have been found in 62 per cent of a survey series of 100 unselected patients with pulmonary tuberculosis presented as candidates for thoracic surgery. Average deficiencies found were 12 per cent in blood volume, 19 per cent in hemoglobin mass, and 8 per cent in plasma volume.

The major deficiency was in hemoglobin mass and exceeded the hemoglobin equivalent of 500 c.c. of whole blood in 62 per cent of the total series; 1,000 c.c. in 46 per cent of the series; 1,500 c.c. of blood in 23 per cent; and 2,000 c.c. in 8 per cent of the entire group.

Blood volume determination is the best method available for evaluating volumetric deficiencies and should be employed in chronic pulmonary tuberculosis in order to provide better supportive therapy in both the medical and surgical phases of treatment.

A discussion of the problems associated with volumetric blood replacement therapy is presented.

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DISCUSSION

DR. LEO ELOESSER, San Francisco, Calif.—It is difficult for me and perhaps for others to follow Dr. Kehne in mathematical considerations involved in these studies. Might I ask him please to explain to us why he selects as a modality the relation of the blood volume to the body surface rather than the body weight; or, indeed, why he selects that modality at all in preference to just an ordinary hemoglobin percentage determination.

DR. KEHNE (Closing).—There are numerous reports in the literature related to the method volumetric expression of blood and plasma volume, and there are some authors who have found that the correlation of blood volume and plasma volume with body weight is best. There are others who maintain that the correlation with body surface, which takes into account both height and weight, gives the best correlation. We have studied about 250 patients and have found that the obesity factor introduces a special problem. In patients weighing 160 pounds or more no correlation could be found between blood volume and weight. It has been suggested that the use of a blood volume-weight correlation based upon the standard weight tables is necessary in the obese patient. In our hands the blood volume-body surface correlation was not entirely satisfactory, but was best in the universal group. The major problem in the calculation of blood volume deficiencies is that it is difficult to establish what constitutes the normal blood volume for a given person unless we have had a preliminary determination in the normal healthy state.

The second question is what advantage the total blood volume method has over the usual laboratory determinations. Slide 2 presents the change we see in the chronically deficient patient. We found an average deficiency of 12 per cent in total blood volume and an 8 per cent deficiency in plasma volume. This implies a changing relationship between the total plasma volume and the total blood volume. Laboratory values for the usual cell counts and hematocrit could be valid only if the plasma volume remained unchanged from the normal state. The failure of the usual laboratory determinations to identify the deficient patient is further demonstrated by the fact that 50 per cent of the deficient patients had normal laboratory values. It has been amply demonstrated in the literature that in the presence of this changing relationship between total blood and plasma volume, the usual laboratory values are invalidated.

VENTILATORY PROBLEMS IN THORACIC ANESTHESIA

A Volume-Cycling Device for Controlled Respiration

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IN THORACIC surgery the anesthesia offers important physiologic and practical problems, the successful solution of which contributes greatly to the total result of the surgical treatment. The institution of controlled respiration in intrathoracic operations has made these operations safer for the patient. It counteracts paradoxical breathing, mediastinal flutter, and the harmful effect of pendulum air. In addition, the operative procedure is greatly facilitated for the surgeon. In spite of this, the establishment of an adequate gaseous exchange is still a major problem in the anesthesia techniques used hitherto.

PHYSIOLOGIC PRINCIPLES OF VENTILATION AND CIRCULATION

In physiologic conditions, the pCO_2 action on the breathing center automatically maintains an adequate alveolar ventilation. During anesthesia with controlled respiration, this sensitive mechanism for regulation of the ventilation is out of action; the responsibility is taken over by the anesthesiologist.

By the administration of a high oxygen percentage in the gas mixture and in the absence of circulatory shunts, arterial hypoxemia is prevented even if there is serious hypoventilation. This is clearly demonstrated in experiments with diffusion respiration.[1, 2] For the proper elimination of CO_2, on the other hand, an adequate alveolar ventilation is necessary. Hypoventilation will result in a respiratory acidosis; hyperventilation in a respiratory alkalosis. Both these conditions, often difficult to recognize without the help of complicated analysis of the arterial blood, are dangerous to the patient. In both conditions the circulation is impaired. If corrective measures are not undertaken, circulatory collapse may supervene.

The gaseous exchange between the lungs and the blood is a diffusion process through the alveolar-capillary membranes. As the CO_2 is highly diffusible through wet membranes, the arterial pCO_2 is the same as the pCO_2 of the alveolar gas to which blood is exposed during its passage through the lung capillaries. For a given patient with a constant CO_2 production, in absence of circulatory shunts, the arterial pCO_2 is a direct function of the magnitude of the alveolar ventilation. Changes in alveolar ventilation will cause changes in the alveolar and arterial pCO_2, inversely proportional to the magnitude of

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the alveolar ventilation. The amount of free carbonic acid in the plasma varies proportionally to its partial pressure. The acid-base balance in the blood plasma is regulated by the buffer system bicarbonate—free carbonic acid. The relationship between the pH and the content of free carbonic acid and bicarbonate in the plasma is expressed in the Henderson-Hasselbalch equation:[3]

$$pH = pK' + \log \frac{BHCO_3}{H_2CO_3} \quad (pK' = \text{a constant } 6.107 \text{ at } 37° \text{ C.}).$$

The total plasma $CO_2 = BHCO_3 + H_2CO_3$. If the total plasma CO_2 is expressed in volumes per cent and the free carbonic acid in mm. Hg, the formula is written:

$$pH = pK' + \log \frac{CO_2 \text{ Vol. } \% - (0.1316 \cdot a \cdot pCO_2)}{0.1316 \cdot a \cdot pCO_2}$$

(a = the solubility coefficient of CO_2 in plasma = 0.521 at 37° C.).

From this equation it is seen that there is a normal pH (7.38–7.41) when the ratio $\frac{BHCO_3}{H_2CO_3}$ is about 20:1, independent of the absolute values of the numerator and the denominator; and inadequate alveolar ventilation will cause a change in the ratio with displacement of the pH. A respiratory acidosis or alkalosis thus occurs. To a certain extent a compensatory retention or elimination of basic ions available for combining with CO_2 to bicarbonate takes place. This may result in a disturbance in the balance of the electrolytes.

The effect on the circulation of pressure breathing in the closed thorax depends on the mean pressures applied to the airways.[4] The importance of a negative phase in pressure breathing to keep the mean pressure on a low level is widely accepted in order to avoid harmful effect on the circulation.[5, 6] A certain fraction of the intrapulmonary pressure is transmitted to the right auricle and the big central veins with a tendency to decrease the venous pressure gradient and thereby even the venous return and the cardiac output. In the presence of normal circulation, this effect is counteracted by a compensatory increase of the peripheral venous pressure, reconstituting the venous gradient. The ability to reconstitute the venous pressure gradient is dependent on normal functioning vasomotor reflexes and the presence of a normal circulating blood volume. In the open thorax, the effect on the circulation of a negative phase and a low mean pressure may be less. The expiratory resistance imposed by a narrow endotracheal tube may lead to an increase in the functional residual air. This can be overcome if the respirator applies negative pressure during expiration. The resultant prevention of excessive lung expansion is often of practical value to the surgeon.

Based on these physiologic principles of ventilation and circulation a method for controlled respiration in thoracic anesthesia has been worked out.

METHOD

First, it is essential to find out the patient's minute ventilation at rest before the operation. It can be calculated fairly accurately[7] and most comfortably by use of a ventilatory nomogram.[8] It is even better with a direct measurement, especially in patients with impaired pulmonary function.

Controlled respiration with the obtained values for ventilation is administrated by a respirator designed by Engström in 1951,[9-11] which is adapted for giving anesthetics by adding a special rotameter attachment for N_2O and O_2 (Figs. 1 and 2). The gas mixture is insufflated into the air passages *in a semi-open system* (no rebreathing). The respirator is different in principle from the usual types of devices for automatic ventilation during anesthesia. *All such*

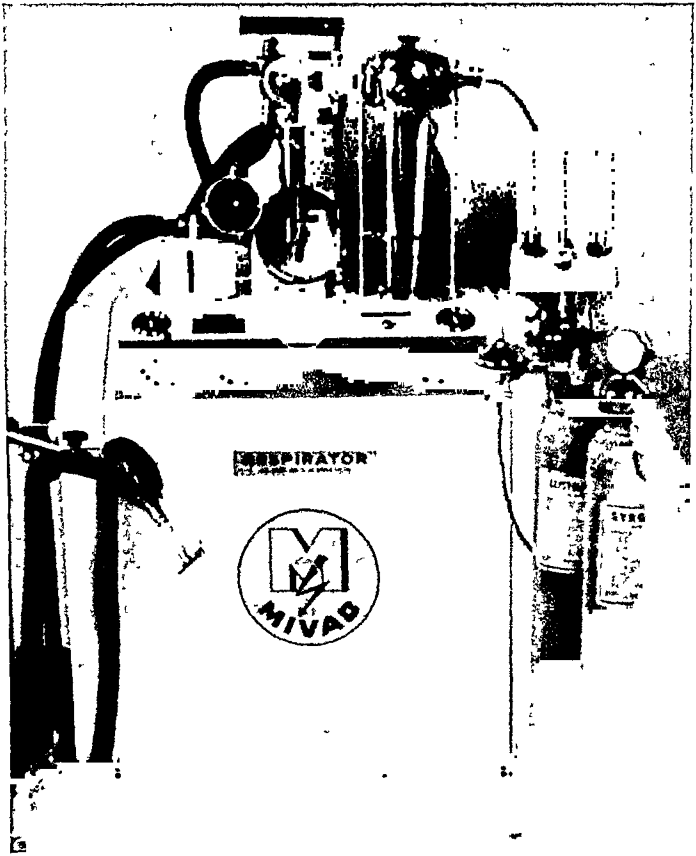
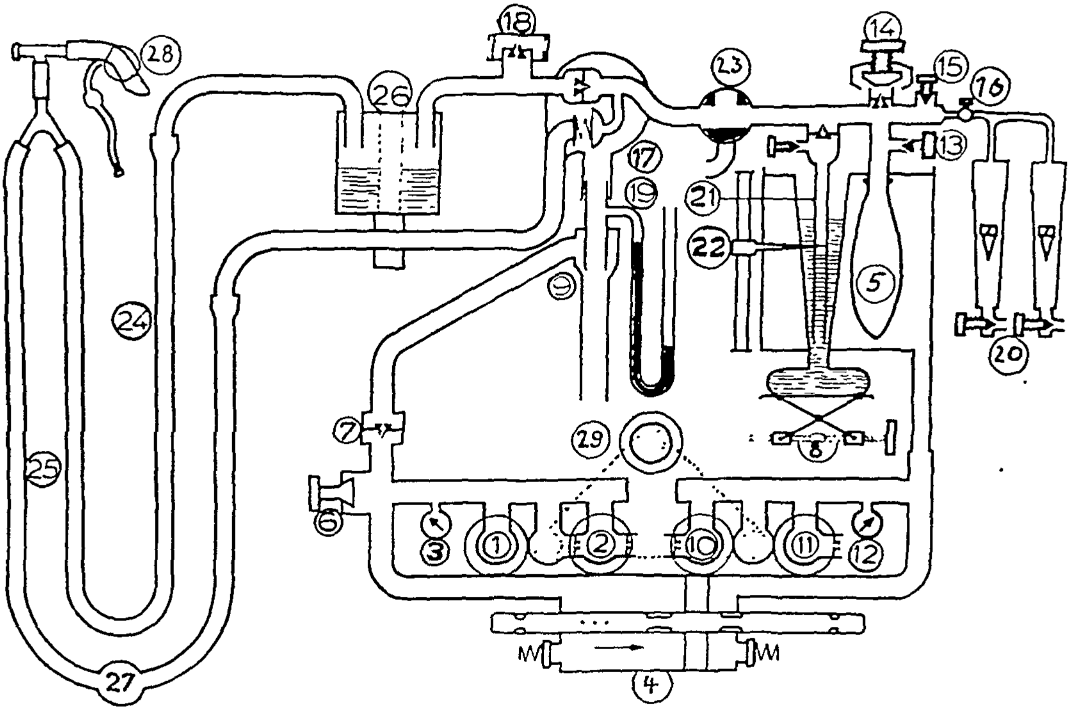


Fig. 1—*For legend see p 120.*

machines in common use are regulated by adjustment of a peak pressure setting and the volume is estimated by the anesthesiologist. This unit, on the other hand, delivers *a pre-set minute volume* at a pre-set respiratory frequency. If the respiratory frequency is changed the pre-set minute volume will not alter. The resultant tidal volume is then calculated from the respiratory frequency and the minute volume. The pressure required to deliver this minute volume will vary from individual to individual; and in the same individual from time to time, since it is determined by the physical characteristics of the lung and chest wall.[12, 13] An increase of the pressure required to force a given volume of gas into the patient's lungs means an alteration of these mechanical factors. *An increased resistance to insufflation will lead to an increased inspiratory pressure and a prolongation of the phase of inspiration.* This will result in a slowing of the rate of gas flow *which will permit the same gas volume to be insufflated with*



Figs. 1 and 2.—The compressor (*4*), which is driven by an electrical A. C. motor, consists of a double cylinder with independent valve systems (*1, 2, 10, 11*) for both pressure chambers. The frequency of the compressor piston is regulated by a stepless mechanical variator (*29*) by which the respiratory frequency can be regulated between 10 and 30 cycles per minute. The pressures from the right part of the compressor are transmitted to a chamber with rigid walls containing a rubber bag. When the piston moves to the left a definite volume of air per time-unit is sucked into the rubber bag (*5*) through an adjustable, calibrated inlet valve (*14*), or the anesthetic gas passes from the rotameters (*20*) into the rubber bag. When the piston moves in the opposite direction the rubber bag is squeezed and the gas insufflated into the air passages of the patient. On the way it passes a waterlock (*21*), a pressure-controlled expiratory valve (*17*), which during the inspiratory phase closes the expiratory passage, and a humidifier (*26*). Before the piston comes to the end of the stroke an automatic pressure release on the piston shaft is opened, which causes a rapid drop in pressure. When the pressures squeezing the rubber bag are set to a higher value than the pressure fluctuation on the manometer in the waterlock the rubber bag will be deflated before the piston comes to the end of the stroke. Thus, there will exist a pressure "reserve" around the outside of the rubber bag, which will be used to overcome any increased resistance in the patient's circuit. The left part of the compressor cylinder supplies the positive pressure fluctuations for operation of the ventury (*9*) which in turn applies an intermittent negative pressure on the atmospheric side of the expiratory valve. This is transmitted to the airway and empties the patient's lungs.

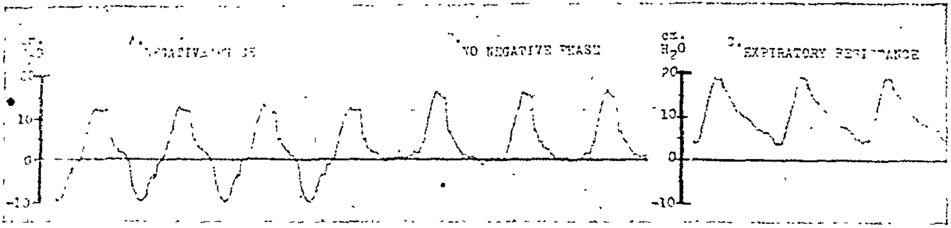


Fig. 3.—Pressure pattern produced by the described device in a narcotized and curarized patient with pulmonary tuberculosis (undergoing lobectomy). The pressures are recorded in the upper part of trachea by closed thorax. Minute ventilation of 9 L. per minute and respiratory frequency of 22 cycles per minute are the same in all tracings.

A, A negative phase of −10 cm. H_2O is applied. Mean pressure = +1 cm. H_2O. *B*, Only positive pressure phase. Note increase of the maximal intratracheal pressure. Mean pressure = +1.3 cm. H_2O. *C*, An expiratory resistance is added. Further increase of the maximal intratracheal pressure. Mean pressure = +9.9 cm. H_2O.

a comparatively smaller pressure increase. This automatic pressure adjustment and adjustment of the rate of gas flow is most important for the successful management of prolonged artificial ventilation in patients with unfavorable factors for the ventilation. The development of dangerous pressures is prevented by a water level safety valve system. Similarly, the negative pressure applied during exsufflation can be regulated. Thus, the mean mask pressure can be kept at a low level (in normal lungs down to zero pressure). By application of a regulating expiratory resistance, it is also possible to operate with high mean mask pressure and with the lungs in expanded position. In spite of this, the patient will get the same amount of ventilation until the safety pressure is reached and gas escapes through the waterlock. Thus, *the mean mask pressure and hereby even the level of the functional residual air can be closely controlled in the presence of a constant minute volume* (Fig. 3). The expiratory volume can be measured to control leakage, if any. The composition of the gas mixture to the patient is exactly known.

COMMENT

Empirically, it is impossible to estimate whether or not the ventilation to a patient under anesthesia is adequate. Several authors have reported severe disturbances in the acid-base balance during thoracic anesthesia.[14-18] As methods for controlling the ventilation by measuring the gas tensions in the arterial blood or alveolar gas are not convenient for practical use, the best method is to control the ventilatory volumes which may serve as a satisfactory index of the adequacy of ventilation.

Different systems may allow volume-recording apparatus (spirometers, pneumotachographs) to be built in. In systems so far used which are pressure controlled, the minute volume will alter according to the alterations in the mechanical factors of the pulmonary ventilation. By use of the described volume-cycling device the patient receives a predetermined ventilartory volume satisfactorily corresponding to his requirements. This is maintained during the operation in spite of variations in the mechanical factors caused by pressure on the lung, traction on the bronchi, and so on.

The described method for controlled respiration has been used in about 70 major intrathoracic interventions in patients most of whom had impaired pulmonary function. By this method it has been possible in 1 patient to keep his ventilation adequate during operation on the only remaining lung. Segmental resection of the left upper lobe was performed. In another patient, both upper lobes were removed in the same stage. Even in "bad risk" patients, the blood pressure and pulse rate have remained remarkably steady during operations lasting for as long as nine hours, indicating that a good circulation has been maintained.

It is often stated that patients in poor condition should receive the highest possible concentration of oxygen in the inspired atmosphere during the operation. Potent anesthetic agents, which allow an oxygen percentage of 80, 90, even 95 per cent, are often advocated. It may be questioned whether this attitude is quite correct. In our opinion, it is preferable to ensure adequate ventilation with sufficient but not excessive oxygen concentration, since then the possibility of hypercapnia will be avoided. Our results in poor-risk patients with N_2O as the anesthetic agent seem to justify such an opinion.

In cases with respiratory insufficiency during the immediate postoperative period after pulmonary resections, the same device is successfully used for artificial respiration.[12]

SUMMARY

For the administration of controlled respiration, a volume-cycling positive-negative pressure device (the Engström Respirator) has been used on a series of about 70 patients undergoing major intrathoracic operations. The method has proved most valuable and offers in the main the following advantages:

1. Dangerous hypo- and hyperventilation is prevented by the administration of a determined ventilatory volume, maintained independently of altered mechanical conditions, such as opening of the pleura, compression of the lung, and traction on the bronchus.
2. Respiration with a negative expiratory phase and low mean pressure favors the circulation, and makes it possible to ventilate the patient when only narrow endotracheal tubes can be used. The degree of expansion of the lungs can be controlled with maintenance of a constant minute volume, and this often facilitates the work of the surgeon.
3. The composition of the gas mixture administered to the patient is exactly known.

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Announcement

New Regulations of the Board of Thoracic Surgery

At a meeting of representatives of the Board of Thoracic Surgery with the American Board of Surgery held on April 26, 1955, in Philadelphia, it was agreed that candidates examined by the American Board of Surgery in Part I (written examination) on or after October 26, 1955, who are certified by the American Board of Surgery, would be required to take only the oral examination given by the Board of Thoracic Surgery.

Before taking the oral examination of the Board of Thoracic Surgery the candidate must furnish satisfactory evidence of having completed three years of training in general surgery and two years of training in thoracic surgery in an approved institution, and that he is a diplomate of the American Board of Surgery.

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Vol. 31 February, 1956 No. 2

Original Communications

SURGICAL TREATMENT IN TUBERCULOSIS COMPLICATED WITH PULMONARY EMPHYSEMA

Robert W. Newman, M.D. (by invitation), Perry M. Huggin, M.D. (by invitation), Charles L. Butler, M.D. (by invitation), and
Medford C. Bowman, M.D. (by invitation)
Knoxville, Tenn.

INTRODUCTION

THE surgical patient with generalized pulmonary emphysema presents a challenging problem in therapeutic management regardless of the nature of the proposed surgical procedure. In the patient with tuberculosis, complicated by the presence of generalized pulmonary emphysema, the need for objective thinking before electing to proceed with a needed surgical procedure is self-evident.[1, 2] This need stems from the current concept that, following long-term drug therapy and other methods of medical management, residual large foci of tuberculosis, whether air-bearing cavities or inspissated cavities or destroyed segments, lobes, or lungs, should be surgically treated; and in most quarters surgical treatment has meant surgical excision.[3-7] Thus, certain patients with tuberculosis, complicated with pulmonary emphysema, become candidates for major excisional or surgical collapse measures in the face of a deranged ventilatory and perfusion system.

We have followed the concept that, following adequate and for the most part prolonged (six to twenty-four months) medical management, the foci which the treatment had not been able to bring successfully under control due to mechanical problems[8] (i.e., bronchostenosis, bronchiectasis, residual cavities either air-bearing or solid) *should be treated surgically. Following the lead of* pioneers in the field, we became convinced early that resectional surgery offered better opportunities for correcting the surgical pathology and at the same time conserving pulmonary function at the highest possible level. Thus, inevitably, we began to accept patients for excisional therapy who had moderate to marked impairment of the pulmonary function prior to surgery due to pulmonary emphysema, regardless of the etiology. It followed that the problems concerned with management of these cases, during the operative and postoperative periods,

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From the East Tennessee Tuberculosis Hospital, Knoxville, Tenn.

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 24 to 26, 1955.

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KNOXVILLE, TENN.

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THE surgical patient with generalized pulmonary emphysema presents a challenging problem in therapeutic management regardless of the nature of the proposed surgical procedure. In the patient with tuberculosis, complicated by the presence of generalized pulmonary emphysema, the need for objective thinking before electing to proceed with a needed surgical procedure is self evident.[1, 2] This need stems from the current concept that, following long-term drug therapy and other methods of medical management, residual large foci of tuberculosis, whether air-bearing cavities or inspissated cavities or destroyed segments, lobes, or lungs, should be surgically treated; and in most quarters surgical treatment has meant surgical excision.[3-7] Thus, certain patients with tuberculosis, complicated with pulmonary emphysema, become candidates for major excisional or surgical collapse measures in the face of a deranged ventilatory and perfusion system.

We have followed the concept that, following adequate and for the most part prolonged (six to twenty-four months) medical management, the foci which the treatment had not been able to bring successfully under control due to mechanical problems[8] (i.e., *bronchostenosis, bronchiectasis,* residual cavities either air-bearing or solid) should be treated surgically. Following the lead of pioneers in the field, we became convinced early that resectional surgery offered better opportunities for correcting the surgical pathology and at the same time conserving pulmonary function at the highest possible level. Thus, inevitably, we began to accept patients for excisional therapy who had moderate to marked impairment of the pulmonary function prior to surgery due to pulmonary emphysema, regardless of the etiology. It followed that the problems concerned with management of these cases, during the operative and postoperative periods,

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soon became acutely real. Furthermore, it is anticipated that the number of patients with tuberculosis, complicated with emphysema, will increase as the trend in hospital admissions throughout the country is toward the older group of patients in whom the incidence of generalized emphysema is much higher. It is in this complicated group of older individuals that surgical therapy is usually mandatory if sputum conversion and arrest of the tuberculosis are to be accomplished.

Our presentation is an analysis of 30 surgical patients with tuberculosis complicated with moderate to far-advanced stages of pulmonary emphysema compared to a parallel group of 259 surgical patients treated during the same three-year period who had pulmonary tuberculosis without a significant degree of complicating emphysema. The complications and morbidity in the emphysematous group have been most impressive and prompted the undertaking of the study. This group also pointed to the need for a critical evaluation of the emphysematous patient prior to surgery and a keener appreciation of postoperative management directed toward the basic physiologic and pathologic deviations.

PATHOLOGY AND PATHOGENESIS

A. The basic tuberculous process takes place in the alveolar spaces, best seen in the acinous nodose foci developing beyond the terminal bronchiole.[9] Healing occurs by invasion of the fibrinocellular exudate in the alveoli by fibroblast, epithelioid, Langhans' cells, and collagen fibers. These elements replace the exudate; the collagen increases and an area of fibrosis develops which is surrounded by torn and stretched alveolar septa. As the granulation tissue is gradually replaced by connective tissue there is a gradual contraction of the focus. As the scar contracts it stretches the surrounding alveolar septa leading to perifocal emphysema.

Emphysema in pulmonary tuberculosis is a concomitant part of the destruction and resultant contraction of the lung parenchyma. As the tuberculous process progresses with necrosis and surrounding fibrosis, a compensatory dilatation of the surrounding air sacs develops. Since the parenchyma is a contiguous elastic structure, any replacement of it must be compensated for by a surrounding intact portion. This is done by stretching of the elastic fibers.

After chemotherapy, there is an over-all decrease in the amount of fibrosis and emphysema in patients who have received chemotherapy over a long period of time.[10] This is true if the drugs are given soon after the onset of the illness. The decrease is due to the elimination of the perifocal reaction resulting in decreased fibrosis and to the resultant decrease in dissemination. As a result of the greatly lessened fibrosis and emphysema in prolonged chemotherapy there is a greater normal lung volume. Intact lung parenchyma, which rarely contains scars, widely separates necrotic foci.

The bronchus communicating with a cavity almost always reveals the same ulcerative tuberculous process as that in the cavity.[9] The bronchocavity junction is a tangential canal resulting in a check-valve mechanism at this site. More air enters the cavity during inspiration than leaves during expiration, usually accounting for the spherical appearance.

Streptomycin treatment causes re-epithelization of the bronchus at the bronchocavity junction with extension along the inner wall of the cavity. This disrupts the check-valve mechanism allowing cavity healing to occur with the end result of an inspissated cavity with a patent communicating bronchus. "Open" healing is observed with increasing frequency after long-term chemotherapy, probably due to re-epithelization of the bronchus.

As a result of prolonged chemotherapy in pulmonary tuberculosis soon after its origin, the disease undergoes extensive resolution and the process is limited to a portion of a lobe or lung. The portion of residual diseased lung can then be removed surgically.

B. Obstructive emphysema in pulmonary tissue may be localized in a segment or lobe or it may be generalized throughout both lungs. It is brought about by an increased tension of the air within alveoli due to a partial occlusion of a bronchus or to a generalized increased air tension due to a partial occlusion of the respiratory bronchioles. The mechanism of the production by air-trapping in the alveolus by obstruction to the outflow of air is well known.[11]

The anatomy of the bronchial musculature indicates smooth muscle and elastic fiber projections to form sphincters about the atrial openings from the respiratory bronchioles.[12] Thus, trapping of air in the alveoli could result from sphincteric closure with outflow obstruction. Cough derived from any cause forces air into the alveoli under pressure which in generalized bronchospastic states or in localized segmental or lobular bronchitis produces an outflow obstruction. If this situation continues it will lead to alveolar overdistention, loss of elasticity in the bronchiolar walls, and permanent changes recognized as pulmonary emphysema. The emphysema may be localized or generalized.

Collateral air-drift as a safety device to prevent overdistention in a lobule or segment remains operative in most instances. However, in some cases it fails to keep the lobule deflated and bullae develop.[13] The process of obstructive emphysema is reversible unless it is prolonged and damage occurs to the elastic tissue in the alveoli; then the emphysema is permanent as the elastic tissue fails to recoil after stretching. In addition, alveolar septa become destroyed, leading finally to a gross cystic structure attached to one or several bronchi or bronchioles. The pulmonary vessels to the cystic area are obliterated. If diffuse emphysema is present in both lungs, peripheral resistance to the outflow tract from the right heart is increased and may lead to cor pulmonale. We have been impressed in our emphysema surgical cases with the markedly increased bronchial artery and bronchial vein blood flow; collateral circulation in both these systemic channels has been noticeable. Liebow and co-workers[14, 15] have written brilliantly on these collaterals and have further described sizable anastomoses between the bronchial and pulmonary arterial systems which represent points of peripheral resistance within the lung and, when sufficiently numerous, increase the work of the right ventricle. In addition, sizable shunts between the bronchial and pulmonary veins exist in emphysema. The exact mechanism is not known. One proposed mechanism would be due, according to the opinion of Loeschcke quoted by Liebow, to the fact that the bullae of emphysema represent expanded portions of the lung tree more proximal than alveoli and include respiratory bronchioles. Then, the venous drainage of the cystic structures, as well as the arterial supply, is systemic in nature rather than from the lesser circulation. Another factor favoring augmentation of the bronchial arterial and venule blood flow is the loss of blood-pumping action from the alternating respiratory pressure changes as they affect the small vessels of a normally elastic parenchyma. This will tend to abolish the differential negative pressure, occurring with each inspiration, which ordinarily favors the flow of blood into pulmonary venules rather than bronchial venules.

The frequent anatomic location of bullae (or "cysts") in the apical segment of the upper lobe and in the superior segment of the lower lobe coincides with the frequent lobar apical localization of tuberculous cavities, which fact leads to interesting speculative possibilities as to the pathogenesis of tuberculous cavities; and further to their frequent failure to respond in emphysematous patients to conservative medical management by cavity closure.

In emphysema, the loss of lung elasticity greatly alters the efficiency of ventilatory function. Respiratory efficiency is decreased because of: (1) The overstretched inelastic subpleural alveoli cannot transmit the pull of the chest wall and diaphragm musculature to the underlying alveoli deeper toward the hilum. This leads to an increase in volume at the tidal expiratory level. (2) Increased ventilatory effort is required to maintain exchange in large lung volume. (3) The diaphragm is depressed in the larger lung volume. (4) The tidal air is unfavorably diluted by the increased residual volume.

Thus, in an emphysematous patient, the residual air may be as large as 50 to 60 per cent of the total lung volume, which leads to dilution of the tidal air.[16] The element of bronchospasm further reduces an already lowered maximum breathing capacity. Carbon dioxide retention is increased and oxygen tension at the alveolocapillary membrane is reduced.

CASE DATA

Our clinical material consists of 289 cases treated surgically for pulmonary tuberculosis. The cases are divided into 2 groups, the first group of 30 cases of tuberculosis, complicated with moderate to far-advanced pulmonary emphysema, and the second group of 259 cases of tuberculosis without a significant clinical degree of pulmonary emphysema.

The patients with clinical pulmonary emphysema are divided into a group with predominant obstructive emphysema and a group with predominant compensatory emphysema. The nonemphysematous group failed to show a significant degree of clinical emphysema changes although, undoubtedly in most cases of moderate and far-advanced tuberculosis, compensatory overdistention occurred as healing progressed. In the 30 emphysematous patients, 26 showed a predominant obstructive complex and 4 showed a predominant compensatory complex of marked degree.

The diagnosis of emphysema was established by: (1) History of "asthma" or asthmatoid wheezing. (2) X-ray evidence of increased radiolucency; depressed and flattened diaphragms; increased anterior-posterior diameter of chest. (3) Fluoroscopic evidence of limitation of diaphragmatic excursion and failure of the lung alternately to lighten and darken with the respiratory cycle. (4) Bronchoscopic examination evidence of a nonspecific bronchitis and bronchospasm. (5) Bronchography was used to outline areas of compensatory emphysema. The distribution of the medium is striking in obstructive emphysema. (6) Ventilatory function studies were used in evaluation and diagnosis. Percentage timed vital capacity is the most helpful in making the diagnosis of the person with obstructive emphysema. The maximum breathing capacity is used in evaluation of the patient's ability to tolerate the surgical procedure. (7) An electrocardiogram which revealed right axis deviation. (8) The operative findings at the time of thoracotomy and pathologic examination of resected specimens were used to confirm the preoperative clinical impression.

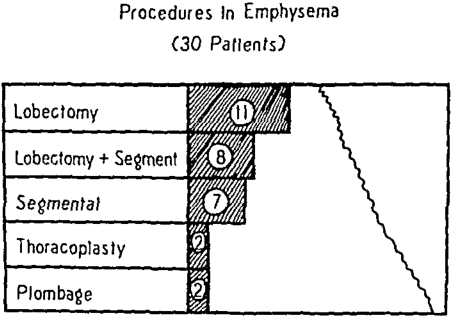

FIG. 1.

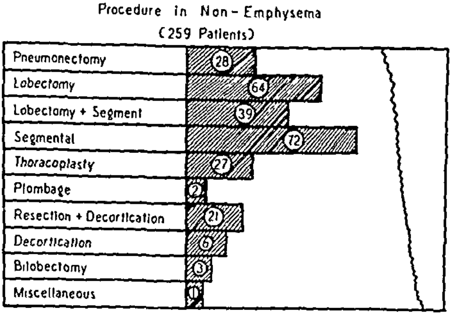

FIG. 2.

The patients were all treated medically with supervised bed rest and long-term antimicrobial therapy. Selected patients were treated with temporary collapse measures such as pneumoperitoneum and, occasionally, with pneumothorax. The preoperative treatment period ranged from six to twenty-four months. Postoperative treatment was continued in most instances from emphysema represent expanded portions of the lung tree more proximal than alveoli and include respiratory bronchioles. Then, the venous drainage of the cystic structures, as well as the arterial supply, is systemic in nature rather than from the lesser circulation. Another factor favoring augmentation of the bronchial arterial and venule blood flow is the loss of blood-pumping action from the alternating respiratory pressure changes as they affect the small vessels of a normally elastic parenchyma. This will tend to abolish the differential negative pressure, occurring with each inspiration, which ordinarily favors the flow of blood into pulmonary venules rather than bronchial venules.

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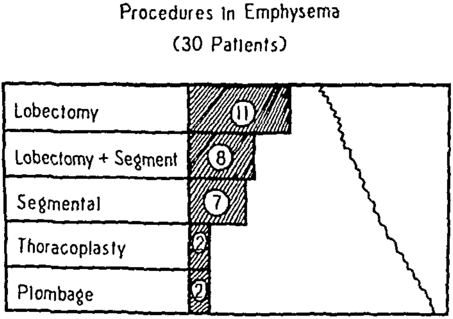

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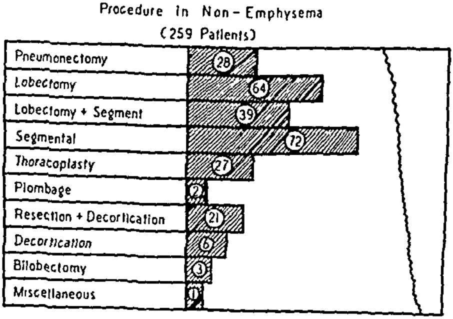

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The patients were all treated medically with supervised bed rest and long-term antimicrobial therapy. Selected *patients were treated with temporary* collapse measures such as pneumoperitoneum *and, occasionally, with pneumothorax.* The preoperative treatment period ranged from six to twenty-four months. Postoperative treatment was continued in most instances from six to twelve months. Postoperative follow-up has been most satisfactory in 96.2 per cent of the cases. Of the emphysematous group, 26.6 per cent had a positive sputum prior to surgery whereas 22.4 per cent of the nonemphysematous group failed to convert to negative sputum before surgery.

The operative procedure employed in the 2 groups of patients are shown in Figs. 1 and 2.

The complications in the 2 groups are shown in Table I.

TABLE I. COMPLICATIONS IN BOTH GROUPS

WITH EMPHYSEMA (%)	COMPLICATION	WITHOUT EMPHYSEMA (%)
57.6	BPF*—delayed re-expansion, no empyema	6.1
19.2	BPF—empyema	3.9
0.0	Empyema—no fistula	1.7
6.6	Spread	3.4
23.3	Miscellaneous	5.0
10.0	Cor pulmonale	0.0
19.2	Residual space	19.6
34.6	Secondary surgery	15.0
23.3	Status—active (31)	11.9†
6.6	(2) Deaths (8)	3.1

*Bronchopleural fistula.

†Included as active are 10 cases less than six months postoperative.

TABLE II. DEATHS IN GROUP WITH EMPHYSEMA

CASE NUMBER	AGE	VENTILATORY FUNCTION	PROCEDURE	CAUSE OF DEATH
415	WM-45	*VC-4,000 L. (%) 55—1 sec. 60—2 sec. 97—3 sec. †MBC-71 L./min.	Preliminary TP‡ stage RUL and Superior Segment RLL	BPF—empyema; death 6 weeks postoperative; hemorrhage; intrapleural
824	WM-43	VC-3,200 L. (%) 56—1 sec. 65—2 sec. 81—3 sec. MBC-63 L./min.	Preliminary TP stage RUL and Superior Segment RLL	BPF—empyema; cor pulmonale; pulmonary insufficiency; death 6 months postoperative

*VC = vital capacity.

†MBC = maximum breathing capacity.

‡TP = thoracoplasty.

The complications in the group of 30 cases of emphysema were numerous and in some instances multiple. In 8 cases, there were no complications and in this group were 4 patients having extrapleural operations. Twenty-two patients out of 26 cases of resection had thirty-seven serious complications with two deaths, a mortality rate of 6.6 per cent. Nine patients required major secondary surgery to correct the complications and 4 required prolonged chest drainage before eventually recovering. In the surviving group, 7 patients are alive but with active disease or with serious residual complications.

On the other hand, the surgical results in the group of 259 cases without serious emphysema have been most satisfactory. Bronchopleural fistula with

TABLE III. DEATHS IN GROUP WITHOUT EMPHYSEMA

CASE NUMBER	AGE	VENTILATORY FUNCTION	PROCEDURE	CAUSE OF DEATH
131	CF-40	VC-1,000 L.	Pneumonectomy	Cardiac failure, 6 months postoperative
151	WF-35	VC-1,000 L.	Pneumonectomy	Pulmonary insufficiency and cardiac failure, 2 weeks postoperative
271	WF-38	VC-1,400 L.	Pneumonectomy	Pulmonary insufficiency and cardiac failure, 8 days postoperative
510	WF-35	VC-1,300 L. (%) 46—1 sec. 69—2 sec. 84—3 sec. MBC-24 L./min.	Monaldi, 1 year; thoracoplasty I; pneumonectomy	Pulmonary insufficiency, 2 days postoperative
213	WF-40	No studies	RUL	Anesthesia death; cardiac arrest at conclusion of procedure
961	WF-32	VC 1,450 L. (%) 62—1 sec. 82—2 sec. 89—3 sec. MBC-27 L./min.	RUL and segment	Air embolism during transfusion, 3 days postoperative
93	CM-28	VC-1,900 L.	Thoracoplasty	Shock during surgery
356	WM-41	VC-1,900 L. (%) 73—1 sec. 82—2 sec. 100—3 sec. MBC-38 L./min.	Decortication	Died 3 days postoperative; pulmonary insufficiency, intrapleural hemorrhage, shock

empyema was present in 3.9 per cent and bronchopleural fistula without empyema (air-leak over seven days and with delayed re-expansion) in 6.1 per cent. Secondary surgery was performed in 15 per cent. The mortality rate was 3.1 per cent. These patients, now well and classified inactive, number 228 or 88 per cent. (Included as active are 10 cases yet in the hospital having been operated upon less than six months ago.)

EVALUATION OF THE PATIENT WITH EMPHYSEMA AND AN ESTIMATION OF HIS ABILITY TO TOLERATE SURGERY

Modern surgical philosophy[17] in the treatment of tuberculosis dictates not only a correction of the inflammatory process but the preservation of pulmonary function at the highest possible level. The mortality rate for resection and surgical collapse is now so favorable that a good result is no longer gauged by inactivation of the tuberculous process; in addition, cardiopulmonary reserves should not be compromised. If severe postoperative complications are encountered, these criteria will not be fulfilled.

Diffuse airway obstruction, as is commonly found in obstructive emphysema and in severe compensatory emphysema, is the chief cause of operative and postoperative difficulty. The complications which occur in this situation seriously impair the cardiopulmonary reserves. It is important that obstructive and compensatory emphysema of a moderate or severe degree be recognized prior to surgery and a surgical procedure selected and operatively managed to assure arrest of the tuberculosis and, also, preservation of respiratory function

Evaluation of the surgical candidate is based on clinical study and certain simple ventilatory function tests in all cases. In selected individuals, special tests are performed. Clinical study includes the usual history and physical examination. Careful attention is paid to a history of wheezing or of "asthmatic attacks," as well as x-ray evidence of a depressed and deformed diaphragm, of increased radiolucency of the lung fields, of widening of the intercostal spaces, and of increased anterior-posterior diameter of the chest on the lateral x-ray projection. Fluoroscopic observation is done with particular attention to the degree of filling and emptying of the lungs, diaphragmatic movements, regional or diffuse airway obstruction, abnormalities of the cardiac shadow, and any abnormalities in cardiac action.

A simple ventilatory test found to be useful in our hands is the maximum breathing capacity which measures the ability of the patient to pump air in and out of the lungs. Several factors enter into the patient's ability to perform this test; first, the neuromuscular function of the chest wall and diaphragm; second, the ability of the lung to distend and collapse; and third, the patency of the airway. Impairment of any of these factors leads to a lowering of the maximum breathing capacity. The other most important over-all function test, especially significant in obstructive emphysema, is the percentage timed vital capacity three-second test. In our hands, it has been most reliable in distinguishing the patient with a high vital capacity and a good maximum breathing capacity who has a large residual volume due to obstructive emphysema.

TABLE IV. EVALUATION OF PATIENTS FOR SURGERY; FUNCTION STUDIES

1. Vital capacity; calculation of per cent predicted
2. Maximum breathing capacity; calculation of predicted
3. Timed vital capacity; per cent exhaled during 1, 2, 3 seconds
4. Walking ventilation
5. Calculation of walking index. $WI = 100 \times WV/MBC$
6. Fluoroscopic examination
7. Bronchoscopic examination
8. Bronchography
9. Bronchospirometry (selected cases) differential (per cent O_2 uptake, ventilation, and VC)
10. Electrocardiogram

Evidence of carbon dioxide retention is important but it can be assumed that the person with diffuse obstructive emphysema is either retaining CO_2 or is unable to rid his blood normally of CO_2 due to the dilution factor at the alveolocapillary membrane. We have not done gas analyses on our patients.

Bronchoscopy is used in all our cases preparatory to treatment and also for surgical evaluation. Evidence of diffuse or regional bronchitis, severe bronchospasm, or bronchostenosis are all recognized and assist in surgical and treatment decisions. Bronchography is most useful in evaluation and diagnosis of the compensatory or obstructive emphysema patient. It is used primarily to outline areas of bronchiectasis or bronchostenosis.

Bronchospirometry is reserved for patients with altered function in whom it is desirable to know to what extent each lung participates in ventilation and gas exchange.

The knowledge of the degree which lungs resist distention and collapse as ascertained by the MBC (maximum breathing capacity) is important when considering an emphysema patient with a "flail chest" due to extensive decostalization procedures. Airway obstruction of emphysema causes the flaccid chest wall to move in further with each inspiration and move out further with each expiration.

Diffuse airway obstruction of pulmonary emphysema or severe compensatory emphysema interferes with proper ventilatory exchange in the alveoli and leads to hypoxia and also to CO_2 retention and respiratory acidosis. The development of respiratory acidosis and cardiac arrhythmias, during operation and in the postoperative period, is recognized and is especially prone to develop in patients with severe ventilatory derangement.[18]

THE "EMPHYSEMA SYNDROME"—A POSTOPERATIVE PROBLEM

The surgical patient with pulmonary emphysema presents a striking, characteristic clinical picture in the postoperative period. The condition is best described as the "emphysema syndrome," as it is a physiologic and pathologic alteration brought on by profound changes in the body mechanism.

Dehydration is a prominent sign and is manifested by poor tissue turgor, dry skin, dry, beefy-red tongue, and oliguria. It is present despite what appears to be an adequate water intake. It is related chiefly to an increased insensible loss of water through the respiratory system related to the hyperventilation of emphysema. Dehydration is prominently associated with respiratory acidosis.

TABLE V. "EMPHYSEMA SYNDROME"

1. Dehydration
2. Hyperventilation
3. Respiratory acidosis; alteration in blood CO_2, sodium, and potassium
4. Toxicity due to retained bronchial secretions
5. Marked paradoxical movement in thoracoplasty cases
6. Protracted air leak in resection cases
7. Intestinal distention
8. Disorientation and hallucinations

Hyperventilation is the only physiologic method that the severe emphysematous patient has to compensate for his inadequate alveolar ventilation and perfusion. Chronic arterial anoxia and carbon dioxide must be combatted and an increased depth and rate of respiration is his only recourse.

Respiratory acidosis may be present in this symptom complex. Hyperpnea and dyspnea with mild exertion are the only fairly constant signs associated with a nondiabetic acidosis.[19] However, when the acidosis is severe breathing may be normal even though the plasma pH is very acid. Tachycardia and headache are inconstant signs. Respiratory acidosis may be present and the only sign be a dyspnea out of proportion to the work being performed. Very severe respiratory acidosis is attended by signs of dying such as weakness, disorientation, stupor, and coma. The CO_2 combining power of the plasma may be elevated as a compensatory reaction to a long-standing CO_2 acidosis as measured preoperatively and be not at all indicative of an alkalosis. Potassium deficits tend to complicate severe extracellular fluid volume deficiency. Alkalosis or acidosis accelerates the inception of potassium deficit for reasons not well understood.

Signs of toxicity with moderate fever, tachycardia, and leukocytosis are related to retained bronchial secretions and lobar or segmental atelectasis. The cleansing mechanism of the bronchus in emphysema is markedly interfered with by the elongation and fixation of the trachea and large bronchi. Ciliary action and bronchial peristalsis are inefficient. Cough of an effective nature depends on movable air and, in emphysema cough is ineffective due to the marked increase in residual air volume. In addition, acute atelectasis contributes markedly to the degree of arterial unsaturation due to venous admixing from the perfused but nonventilated lung.[20]

The marked paradoxical movement in thoracoplasty cases with the resultant poor air exchange is self-explanatory. Protracted air leak from the raw surface of the lung in segmental resections and from undeveloped or fused interlobar fissures has been a major problem. The continued air leak necessitates prolonged tube drainage with the increased likelihood of pleural contamination.

Intestinal distention of moderate to major proportions has been evident. One factor is probably due to swallowed air in the hyperventilating and anoxemic individuals, and a second is the intestinal distention that accompanies hypokalemia. A third factor is related to the abdominal type respiration which most of these emphysema patients depend upon, especially in those with fixed thoracic cages.

Disorientation and hallucinations are seen frequently and are prone to develop in the patient with respiratory acidosis of a moderate or severe degree—these are usually late signs and indicate marked metabolic derangements.

CLINICAL APPLICATION OF SOLUTION TO PROBLEM AS DERIVED FROM OUR EXPERIENCES

A. *Selection of Operative Procedure.*—Numerous previous discussions attest to the divergent opinions with regard to the surgical treatment of pulmonary emphysema per se and also as to the management of overdistention of the residual lung or lung tissues following pulmonary resection. It has been pointed out that a postoperative reactivation or spread of tuberculosis in the emphysematous lung causes the patient to do poorly and usually to an extent proportional to the degree of emphysema.[10] From our studies, we would conclude that the patient with tuberculosis and diffuse obstructive pulmonary emphysema does poorly with resectional surgery in a high percentage of cases. Resectional surgery in the patient with compensatory emphysema of even a marked degree is associated with fairly good results. An upper preliminary stage of thoracoplasty of two or three ribs prior to the resection helps to ensure eradication of any space problems and assists in overcoming persistent air leaks. If a bronchopleural fistula and empyema develop, the resultant space will be small. Sufficient interval should elapse between the stage of thoracoplasty and the resection so that a relatively stable chest wall will be present postresection, otherwise, paradoxical movement of the chest wall will negate the value of the preliminary thoracoplasty.

TABLE VI. SELECTION OF OPERATIVE PROCEDURE

1. Preresection thoracoplasty (small stages)
2. Monaldi tube—long-time suction followed by thoracoplasty
3. Extraperiosteal plombage
4. Primary resection—especially in cases of compensatory emphysema

The value of Monaldi catheter suction in obliterating large, tension tuberculous cavities is time-proved[21] and in the patient with associated obstructive emphysema it can be life saving. The long-term catheter suction treatment should be followed by an extrapleural thoracoplasty to ensure permanent collapse of the diseased area. Perhaps the idea of converting the sputum to negative will not be realized in all cases but more patients will be alive to have sputum.

Extraperiosteal plombage is an excellent alternative method in selected cases. The function-sparing aspects of this operation have been repeatedly shown. It is particularly useful in apical, bilateral, cavitary disease. The emphysematous patient tolerates the procedure well. The disadvantage of inserting a foreign material in the body is recognized, but with careful technique only a few will become complicated, and if so the prosthesis can be removed and a thoracoplasty added to obliterate the resultant space.

The combination of a lowered maximum breathing capacity and of a low percentage timed vital capacity in a patient with obstructive emphysema is a very poor prognostic sign and would serve to indicate that resectional surgery is contraindicated. Our results would indicate that these patients tolerate the operative procedure very well but the high percentage of postoperative complications, led by bronchopleural fistula, tend to cause postoperative derangement which is insurmountable. On the other hand, a patient with a good maximum breathing capacity but with a low percentage timed vital capacity will probably have difficulty but will survive, although the complications of resectional surgery will compromise function in the remaining lung tissue.

B. *Operative Management.*—Preoperative treatment should be directed toward relief of bronchospasm with bronchodilator drugs. Expectorant mixtures should be used. Postural drainage and the use of the intermittent positive pressure breathing apparatus with exhalator phase is recommended. Avoidance of cigarettes or cigars is advised. Adequate parenteral fluids and electrolytes should be given to improve the extracellular fluid compartment and to establish the blood CO_2, sodium, and potassium at normal levels. Digitalization in the cor pulmonale cases is indicated. It is desirable to have a negative sputum or at least have drug-sensitive bacilli at the time of operation.

The anesthetic management during surgery is a particularly important phase. It is important that respiration be assisted even to the point of hyperventilation throughout although it is difficult to avoid CO_2 accumulation and resultant acidosis during open-chest operations. Greatly prolonged operating time is to be avoided as there is a direct relationship between time elapsed and degree of CO_2 retention and resultant acidosis. Blood replacement therapy should be exact by the dry-sponge weight calculation method. The choice of agent depends on the judgment in each case, although our choice in bronchospastic states has been a gas-ether-oxygen combination.

We feel that the use of tracheostomy at the time of surgery will do much toward solving some of the postoperative difficulty with the "emphysema syndrome."[22] By eliminating 100 to 150 c.c. of "dead air" space, effective movement of tidal air is facilitated. The dilution factor at the alveolar membrane is reduced and by having ready access to catheter suction of secretions airway cleansing is effective. Any paradoxical movement can be controlled and paradoxical motion is a big problem in obstructive emphysema. Tracheostomy requires constant supervision by trained personnel; effective cough mechanism is lost in intubated patients. If tracheostomy is not done, then frequent use of endotracheal suction or bronchoscopy is indicated to secure airway patency and to prevent atelectasis.

Careful attention to fluid and ion requirements, especially accounting for the insensible water loss due to hyperventilation is necessary.

The usual antimicrobial regime is utilized postoperatively, frequently combined with the broad spectrum antibiotics.

TABLE VII. POSTOPERATIVE MANAGEMENT OF "EMPHYSEMA SYNDROME"

1. Parenteral fluids
2. Correction of CO_2, sodium, and potassium imbalances
3. Tracheostomy (early in selected cases)
4. Bronchoscopy plus catheter suction
5. Bronchodilator drugs
6. Intermittent positive pressure breathing
7. Antibiotics
8. Avoidance of paradoxical motion
9. Sparing use of narcotics
10. Digitalization as indicated

In conclusion, we feel that there is no larger, unsolved problem in the oracic surgical field than the patient with obstructive emphysema. The prob-ns are manifold and his treatment is complicated. When the emphysema is nplicated by tuberculosis (or the tuberculosis complicated by emphysema), becomes a community problem and as such becomes eventually a candidate r surgical treatment. The major points to be considered in the surgical management are (1) the selection of an operative procedure designed to arrest the tuberculosis and conserve pulmonary function at a level compatible with an active but restricted life; (2) preoperative and operative management to correct, in so far as possible, the deranged ventilatory function which has altered the basic mechanism of the body; and (3) prompt recognition of postoperative complications and institution of corrective measures.

SUMMARY

1. The surgical treatment of pulmonary tuberculosis complicated with either severe obstructive or compensatory emphysema is marked by multiple complications in the postoperative period. The complications are particularly severe if resectional surgery is performed.

2 Comparative studies on results obtained in the surgical treatment of 30 patients with tuberculosis and pulmonary emphysema are made with 259 patients with relatively uncomplicated tuberculosis The incidence of bronchopleural fistula with empyema, and persistent air leak without empyema was remarkably high in the emphysema group Bronchopleural fistula with empyema was the complication resulting in the two deaths out of the 30 cases, a mortality rate of 6 6 per cent In the nonemphysema group of 259 cases, eight deaths occurred, a rate of 3 1 per cent, none of which were due to bronchopleural fistula or empyema

3 The "emphysema syndrome" is described and suggestions as to operative management are made

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DISCUSSION

DR. AUSTIN R. GRANT, Brooklyn, N. Y.—In resectional therapy of cases where the remaining lung tissue is emphysematous, a major consideration is to prevent abnormal volumetric changes of the remaining parenchyma. In order to prevent these changes, an intrapleural tailoring procedure, originally devised by Dr. Miscall and Dr. Klopstock in 1947, is now being used and studied by Dr. Klopstock, Dr. Seibens, and me at the Veterans Administration Hospital, Brooklyn, N. Y., in patients with emphysema of the lung and where large segments of lung have been excised.

The method consists of reroofing the pleural cavity with a sheet of parietal pleura following partial pulmonary resection. It is tailored to the configuration of the residual pulmonary parenchyma, thus eliminating any residual space.

It is accomplished without the trauma of rib resection. Importantly, it prevents sudden overdistention of the residual pulmonary parenchyma, allowing a subsequent graded and adaptable rate of pulmonary expansion. The tent minimizes respiratory distress in the patient with marginal pulmonary reserve. It is useful in bilateral procedures and is relatively reversible.

A patient, H. A., with bilateral cystic disease and emphysema had an excision of a left lung cyst with pleural tent on July 24, 1954, and a right upper lobectomy with a pleural tent on Nov. 10, 1954. The preoperative and postoperative roentgenograms are shown.

Postoperative pulmonary fuctional studies reveal a decrease in residual volume, a decrease in vital capacity, an unchanged ratio of residual value to total lung capacity, and an increased maximum breathing capacity. This is a desirable and favorable functional result, avoiding the abnormalities of volumetric relationships which occur in overdistention of lung parenchyma.

A color film shows the technique of reroofing with a pleural tent.

Postoperative roentgenograms of a second patient illustrate the immediate selective limitation of expansion of the residual lung and the subsequent gradual incomplete absorption of fluid and air above the tent.

DR. WILL C. SEALY, Durham, N. C.—Respiratory acidosis is a frequent and difficult problem in the management of emphysematous patients. It is one that we have to deal with not only during operation but in the immediate postoperative period as well.

(slide) This is not a patient with tuberculosis, but one with a spontaneous pneumothorax that has been present for six months, associated with a bronchopleural fistula. This patient had markedly impaired respiratory function.

(slide) This shows a preoperative determination of the carbon dioxide tension of the blood. He had marked ventilatory impairment with a pCO_2 of 65 mm. Hg, and an oxygen saturation of 82 per cent. He had a left thoracotomy with decortication and expansion of the left lung. He did quite well during the procedure, but on the first postoperative day the systolic blood pressure was found to be below 100. He was semicomatose. At that time the pCO_2 had increased to 169 mm. Hg, the pH had dropped to 6.97, and the oxygen saturation increased to 99 per cent. The nasal oxygen administration was stopped. In twelve minutes' time, the pCO_2 dropped to 119 and the oxygen saturation to 89 per cent. This patient made an uneventful recovery, the mental changes cleared completely, and at the time of his discharge the oxygen saturation was back to 95 per cent, and there was only a slight elevation in pCO_2. Patients with chronic respiratory acidosis breathe by means of the hypoxic stimulus to the aortic and carotid bodies; and if this stimulus is removed, they go into marked ventilatory suppression, causing the CO_2 to increase further, followed by coma.

(slide) This shows a preoperative film on a patient with carcinoma of the lung, whose condition was quite good before operation. The next slide shows what happened following surgery.

(slide) The second day after operation he developed expiratory wheezes with bronchial obstruction. On the seventh day he became semicomatose. He was started on ACTH, and in the meantime had frequent aspirations by bronchoscopy. On the ninth day we found the pCO_2 had increased from 67 to 96 mm. Hg. The pH had dropped from 7.38 to 7.16. He was completely comatose. We then did a tracheotomy, put him in a Drinker respirator, and gave him intravenous cortisone. He gradually improved. On the tenth day we removed him from the respirator, and he made an uneventful recovery. At the time he left the hospital he still had chronic respiratory acidosis because of emphysema.

Postoperative respiratory acidosis is a more widely prevalent problem than is generally recognized. It can be easily confused with cardiac failure and cerebral embolus. One should be aware of this possibility when oxygen is administered to postoperative emphysematous patients. Its presence can be proved by estimation of pCO_2 and pH on arterial blood.

DR. NEWMAN, (Closing).—I should like to thank the discussants, and to say a word or two more about operative management. We think that the management of these patients during the operative period is particularly important; it is important that respiration be assisted even to the point of hyperventilation, and at the conclusion of the operation, when the patient has been recognized as being one who is going to have difficulty from what we call the "emphysematous syndrome" postoperatively, we do a tracheotomy at the time, before the patient leaves the table. This, we think, does much toward solving the majority of the postoperative problems, because by eliminating approximately 100 to 150 c.c. of "dead air" space, effective movement of the tidal air is facilitated and the dilution factor at the alveolar membrane is reduced.

In conclusion, then, we feel that there is no larger, unsolved problem in the thoracic surgical field than the patient with obstructive emphysema. The problem is manifold and the treatment is complicated. Emphysema complicated with tuberculosis becomes a community problem, and a patient with such a combination usually becomes a candidate for surgical treatment.

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SURGERY FOR CAVITARY TUBERCULOSIS IN PATIENTS WITH A SINGLE LUNG

FRANCIS M. WOODS, M.D., NORMAN J. WILSON, M.D., AND
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THE management of a segment or lobe destroyed by tuberculosis in the lung remaining after a contralateral pneumonectomy presents a real challenge when it will not respond to antimicrobial treatment. Pneumothorax has been tried. We have attempted cavity drainage, Monaldi drainage, modified thoracoplasties, and extrapleural collapse, usually without favorable issue.

All these procedures significantly reduce the function of the uninvolved segments. Resection of diseased and useless segments or lobes decreases function to a lesser degree if the thoracic cage is opened and then repaired anatomically. The benefits of excisional therapy need not always be withheld from patients possessing only one lung.

In 1951, the successful wedge resection of an isolated metastasis in a single remaining lung was reported by Waterman. Shortly afterward we were asked to help a young pregnant mother with a ballooned-up cavity in the apical segment of the right lower lobe. The left lung had been resected. The diseased segment was surgically excised and she recovered and later delivered normally. The baby is well. The mother is alive, although restricted in her activity. This experience has led us to attempt to salvage 5 additional patients with tuberculous lesions in the lung remaining after a pneumonectomy (Table I). All had failed to respond to prolonged and varied antibacterial therapy.

The surprising feature of the operative procedure has been that in the face-down position the patients were carried through an uneventful anesthesia with respirations only lightly assisted. An endotracheal tube was inserted under topical cocaine. Cyclopropane induction was followed by ether-oxygen maintenance manually assisted when the chest was open. The thoracic cage was entered through an interspace with only one or two ribs divided posteriorly. In closing, the cut ribs were notched, interlocked, and sutured together and the interspace closed thus restoring chest wall contour and stability and motion.

The operative procedure was but slightly hampered by the constant slow rhythmic inflation and deflation of the lung. Only limited resections were undertaken. This involved one subsegmental, four segmental, and two lobar resections. At the close, two intrapleural catheters were used for underwater seal drainage with regulated suction. Postoperatively, particular attention was paid to management of bronchial secretions and maintenance of a good airway.

From the Overholt Thoracic Clinic, Boston, Mass.

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 24 to 26, 1955.

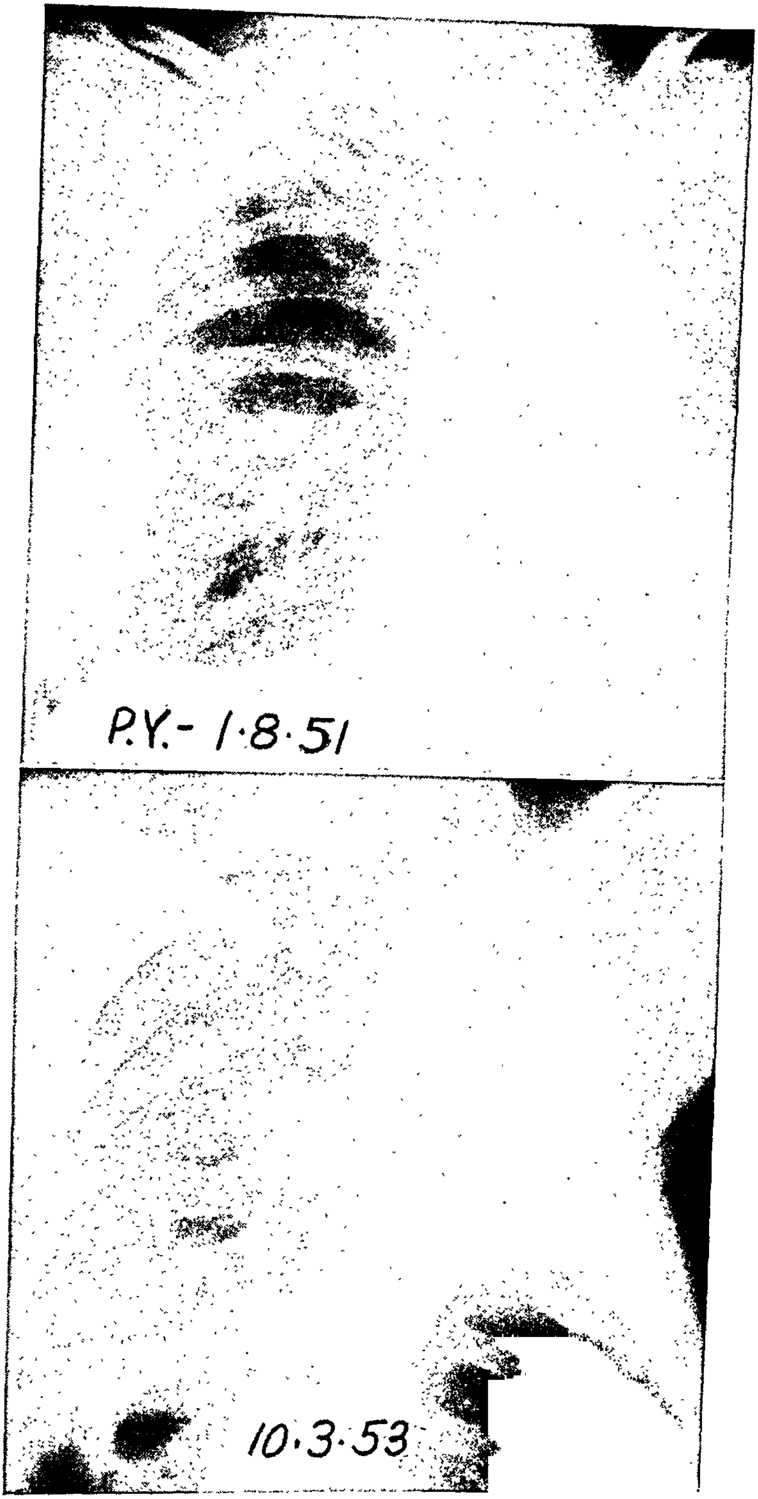
TABLE I CASE SUMMARIES

NAME	AGE	SEX	PNEUMONECTOMY	RESECTION ON REMAINING LUNG	COMPLICATIONS	RESULT (APRIL 1955)
P Y	32	F	Left Mar 30, 1948 Destroyed lung	Superior seg right lower cavity tuberculosis, Jan 19 1951	4 mo pregnancy, bronchoscopy 4th day Spontaneous pneumothorax, 7th day	Alive, sputum positive X ray stable
2 J G	48	M	Left, Jan 21, 1950 Destroyed lung	Post seg right upper fibrocaseous tuberculosis with cavity, Mar 26 1952	Air leak and atelectasis	Death fourth postoperative day
3 D T	37	F	Left June 15, 1949 Destroyed lung bronchostenosis	Sub seg apex right lower lobe 1 cm inspissated cavity Oct 28, 1952	None	Normal life sputum neg
4 F Z	32	F	Left April 4, 1951 Destroyed lung	Right upper lobe, fibrocaseous tuberculosis with cavity, Dec 30, 1952	None	Normal life sputum neg
5 M O'L	41	F	Left June 13, 1951 Destroyed lung, bronchostenosis	Right upper lobectomy fibrocaseous tuberculosis with cavity Nov 12, 1953	None	Normal life sputum neg
6 G C	24	F	Right June 30, 1950 Destroyed lung endobronchial disease	Superior seg left lower lobe [illegible] anterior seg left upper fibrocaseous tuberculosis with cavity Nov 12 [illegible]	None	Normal life sputum neg

Several factors seem worthy of consideration in choosing postpneumonectomy patients who can survive a limited resection in the remaining lung. *First,* age: younger individuals will tolerate greater resections. This group averaged 36 years of age ranging from 24 to 48 years. *Second,* residual disease in the lobes and segments to remain and support [illegible] very little emphysema or nodulation will be tolerated in these segments. The only death occurred in a 48 year old man who had some palpable nodules in all the segments left after resection of the posterior segment of the right upper lobe. *Third,* side: the right lung is larger and therefore, has a larger factor of safety. Five of these six resections were from the remaining right lung. Evaluation of adequacy of pulmonary function was based on clinical examination particularly the ability to perform ordinary activity without dyspnea.

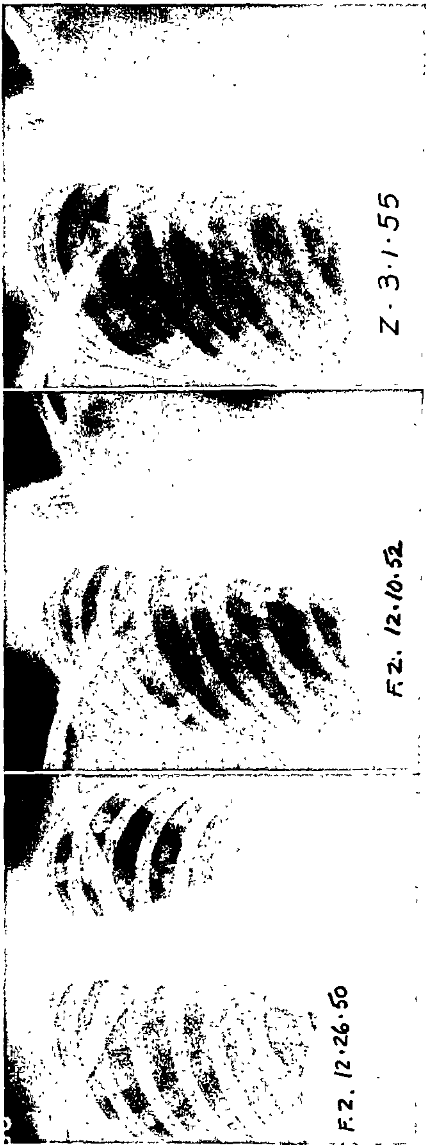
Five of the six apparently hopeless derelicts are alive. Four of them lead normal active lives, have consistently negative sputum and stable chest x rays, one and one half to two and one half years since operation. Their operative and postoperative courses were uneventful. One patient, the first of the six, leads a restricted life and has intermittently positive sputum but stable chest x rays three and one half years later. The one death, already alluded to, was in a patient with nodules in all residual segments. [illegible] There was difficulty in [illegible] of secretions. He [illegible] attaining complete re-expansion of the lung and m[illegible]ation wa[illegible] died on the fourth postoperative day. The only

A.



B.

Fig. 1.—*A* and *B*.



A.

B.

C.

Fig. 2.—A, B, and C.

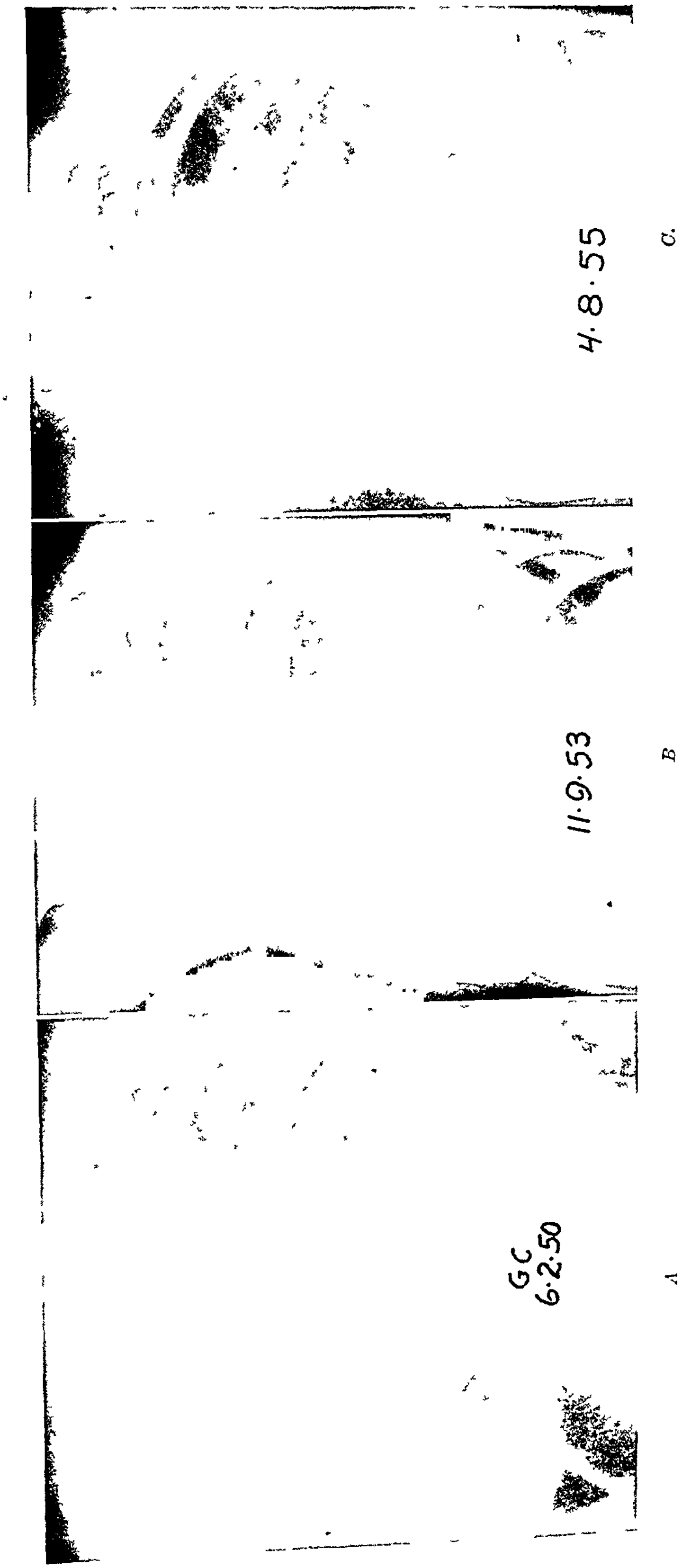


Fig 3.—*A*, *B*, and *C*

taneous pneumothorax occurring on the seventh postoperative day. Fortunately, an alert resident recognized the problem and promptly inserted an intrapleural catheter for underwater seal drainage.

CASE REPORTS

CASE 1. (P. Y.) The patient was a 32-year-old woman who had been treated for 11 years by left pneumothorax, thoracoplasty, and, finally, resection of a destroyed left lung in 1948. She had had several courses of streptomycin. After the pneumonectomy, she had intermittent positive sputum and an infiltrate in the right mid-lung field. On referral to us, she was four months pregnant and there was a cavity in the apex of the right lower lobe (Fig. 1, *A*). A segmental resection of this segment was carried out uneventfully on Jan. 9, 1951. She needed bronchoscopic aspiration on the fourth postoperative day and an intrapleural catheter inserted on the seventh postoperative day for tension pneumothorax. Despite continued and varied antibacterial drugs and pneumoperitoneum, she still has intermittently positive sputum. Her activity is limited to a quiet home life and the chest x-ray remains stable (Fig. 1, *B*).

CASE 4. (F. Z.) The patient was a 32-year-old woman who had been treated primarily for basal left-sided pulmonary tuberculosis since May, 1949, by rest and left phrenic nerve paralysis. In 1950, there was a right-sided spread with pregnancy. Later, treatment by pneumoperitoneum was added but left pneumonectomy and subsegment thoracoplasty were necessary in April, 1951. The right-sided lesion had cleared (Fig. 2, *A*), but later broke down and failed to respond to a series of antibacterial agents. Cavitation persisted. She was clinically well and x-ray study appeared to show all the disease in the apical and posterior segments of the right upper lobe (Fig. 2, *B*). On exploration of the right chest, the entire right upper lobe was found to be involved. It was removed and an uneventful convalescence ensued. She is home, well, active, and only slightly dyspneic on exertion. Chest x-ray remains clear (Fig. 2, *C*) and sputum negative.

CASE 6. (G. C.) The patient was a 24-year-old woman who had been treated for extensive parenchymal and endobronchial tuberculosis of the right lung beginning in May, 1949, with streptomycin, para-aminosalicylic acid, and pneumoperitoneum (Fig. 3, *A*). She had had a right pneumonectomy in June, 1950, and later a thoracoplasty. In January, 1951, there was a left-sided spread. By November, 1953, she was clinically well but had a persistent cavity in the apex of the left lower lobe (Fig. 3, *B*). A segmental resection of this segment was done together with a wedge from the anterior segment of the left upper lobe. No complications occurred and she is well with stable x-ray and negative sputum (April, 1955). (Fig. 3, *C*.)

SUMMARY

Fibrocaseous tuberculosis with cavity existing in the remaining lung after contralateral pneumonectomy which is resistant to antibacterial agents has presented an ordinarily insuperable therapeutic problem. Limited excision is possible, if the remaining segments are healthy. Younger patients will tolerate such excision best, particularly if the remaining lung is on the right. Six such resections have been carried out with 4 unqualified successes, 1 death, and 1 patient alive but with intermittently positive sputum.

Note: We have recently (Feb. 3, 1955) extended this procedure to a 63-year-old white man who had a segmental resection of the apical posterior and anterior segments of the left upper lobe for epidermoid carcinoma. He had had a right pneumonectomy for adenocarcinoma in August, 1950.

DISCUSSION

DR. E. E. ROCKEY, New York, N. Y.—I rise to present the case of a 53-year-old white man who was submitted to segmental resection of the remaining lung on June 1, 1950. He had undergone a left pneumonectomy in February, 1949, for undifferentiated carcinoma.

(Slide) Fifteen months later a recurrence of the disease was noted, which was located in the superior segment of the right lower lobe. The patient was submitted to a resection of the superior segment of this right lower lobe. He made an uneventful recovery and was discharged from the hospital twelve days following the operation. Within six weeks he returned to his usual occupation as a house painter. He remained well for an additional four months.

(Slide) An x-ray taken eight months following the segmental resection showed evidence of recurrence of the disease, and ten months following the segmental resection, a total of twenty-five months from the time of the left pneumonectomy, he died of generalized metastases.

DR. BERT H. COTTON, Los Angeles, Calif.—I rise for three reasons; first, to tell Drs. Francis Woods and Norman Wilson how much I have enjoyed their paper. Characteristic of their attitude and that of Dr. Overholt concerning resection for tuberculosis, they have surmounted technical difficulties to give the patients the best chance for cure. Second, since there seems to be a paucity of pulmonary resection in patients with a single lung, I should like to report 2 successful cases.

(Slide) This is an important decision to make in cases that are otherwise hopeless. However, I have viewed these cases with misgiving since our last case, which presented such a crucial and possibly criticizable decision.

The third reason is to show slides of this desperate case.

(Slide) The patient, a 45-year-old man, had coexisting tuberculosis and coccidioidomycosis with a destroyed left lung and empyema. A left extrapleural pneumonectomy was successfully performed but the patient continued to have hemoptysis with enlargement of the contralateral cavity. A right upper lobectomy, plus resection of the superior segment of the right lower lobe, was necessary to remove the multiple cavitation. The postoperative course was difficult due to fluid accumulation. The patient survived and did well.

DR. DAVID H. WATERMAN, Knoxville, Tenn.—The equanimity with which the papers this morning have been received testifies to the progress of thoracic surgery in the field of tuberculosis, and probably also testifies to the mellowing process that has taken place in many of the more articulate members of this Association. Certainly nothing is sacred. Dr. Woods and Dr. Wilson deserve particular commendation for their contribution.

Encouraged by our success in the case to which Dr. Woods referred, reported before this group in 1949, we have never hesitated to recommend surgical attack on the contralateral lung in pneumonectomized patients when we thought it was indicated. Certainly anesthesia is a very important point as Dr. Woods has mentioned; such surgery cannot be done without very competent and successfully controlled anesthesia. I might say, too, that it is important to cut down the operative time as much as possible, although I doubt if we can perform these operations quite as quickly as Dr. Grant did in his movie. Postoperative care is naturally of exceeding importance, as is the cooperation of the patient. Sometimes I am inclined to think that the patients we see at the State Tuberculosis Hospital and at some of our other sanitoriums are not quite as intelligent as those Dr. John Lewis sees at the State Mental Hospital.

I thought the Association might be interested in a brief follow-up on the patient on whom we reported at the meeting in 1949. This was a 52-year-old man who had had a left pneumonectomy performed in the fall of 1942 for a squamous-cell carcinoma of the left lower lobe. Six years later, in the fall of 1948, we resected an isolated metastasis in the posterior segment of the right upper lobe. His postoperative course was uneventful and he survived for more than two years, during which time he led an active business life. He died eventually of extensive liver metastases.

DR. JAMES E. DAILEY, Houston, Tex.—Despite the apparent ease with which Dr. Waterman shrugs off operating on pneumonectomized patients, it is certain that one thinks with some degree of hesitation about operating on a person with only one lung. Dr. Woods should be congratulated indeed on the nice series of cases that he has presented.

(Slide) I would like to show a case illustrating the number of insults some individuals with only one lung can put up with and still survive. This man, in May, 1950, had a large squamous cell carcinoma of the right lung A little over a year after removing it, I was much surprised to see the right chest cavity filled with a large cyst containing air This turned out to be a huge emphysematous bulla that crept across the anterior mediastinum from the left upper lobe and completely filled the right thoracic cavity It caused no symptoms and the individual went along for a number of years with no trouble whatsoever until December, 1953, over a year ago

(Slide) Suddenly, he developed a spontaneous tension pneumothorax and collapse of the left and only lung Fortunately, I saw him quickly enough to put a tube in his chest re expanded the lung with suction and instilled 10 per cent glucose in the pleural cavity, hoping that pleural symphysis would occur The lung re expanded, he went home, and about a week later the same thing happened a second time Again a tube was placed in the chest and the lung re expanded This time I placed 20 per cent glucose in the pleural cavity He was again discharged to his home and three weeks later the lung collapsed for a third time He was brought back to the hospital cyanotic and gasping for breath and a thoracotomy tube was reinserted This time, since there was no LePage's glue handy, I inserted 50 per cent glucose The lung re expanded and I began to think of something to do to prevent any more such occurrences

Finally, we got courage enough to operate I opened the right chest, removed the tremendous bulla which completely filled the hemithorax, ligating and dividing it from the left upper lobe I then filled the space with an Ivalon sponge prosthesis That was in March 1954 A little more than a year has now passed and the patient has been perfectly well to this date Incidentally, he is a good example of a five year cancer cure

DR RICHARD H OVERHOLT, Boston, Mass—I should like to report one case that is related to the technical problem of those presented by Dr Woods This case is rather unique in that it represents, I think, the only instance in which I sat by for several weeks in the presence of x ray evidence of cancer and watched a shadow grow

The patient was a 63 year old doctor who had been treated for a carcinoma of the right lung by pneumonectomy four and one half years previously In May of last year, an area of density was noted in the apex of the remaining left lung No investigation or treatment was advised I saw him again in November after he had experienced a pulmonary hemorrhage The x ray revealed considerable enlargement of the area of density It now occupied the upper third of the left lung field He came back in January and wanted to know if I would not at least investigate and consider resection Upon bronchoscopy, the left hilar structures were mobile All studies of secretions were negative A cervical node dissection was done and those were negative He then said he was going to commit suicide if we did not stop the bleeding He had had three or four rather frightening hemorrhages I was then persuaded, somewhat against my better judgment, to explore the left chest This was done, and we found the mediastinal nodes to be negative We resected the postero apical and anterior segments which contained the mass A fairly large lingula and lower lobe were all that remained He had a smoother convalescence following this operation than that following the right pneumonectomy He was discharged on the seventeenth postoperative day

(Slide) There is a final interesting point about this case He had an epidermoid carcinoma on the first side and an adenocarcinoma on the other He had been a heavy cigarette smoker We persuaded him to give up the habit four and one half years before Surgery saved him from the cigarette type cancer, and he lived long enough to develop a non smoker's type of cancer

He experiences only slight dyspnea on exertion and is free of all pulmonary symptoms Recently, there was a urinary shutdown which required a transurethral prostatic resection

DR JOSEPH GORDON, Albuquerque, N M—I believe in the relationship between the reactivation of tuberculosis and overdistention of the lung, as was indicated in the paper on pulmonary distention presented two years ago I should like to suggest to those of you who are contemplating a pneumonectomy in tuberculosis, to consider carefully any residual lesions in the contralateral lung and do a pr [illegible]
embarking upon a pneumonectomy on the opposite side

THE SURGICAL TREATMENT OF PULMONARY HISTOPLASMOSIS WITH AN EVALUATION OF MRD-112 AS A POSSIBLE ADJUNCT

JOHN W. POLK, M.D.* (BY INVITATION), CHARLES A. BRASHER, M.D.**
(BY INVITATION), JOAO DECASTRO, M.D.*** (BY INVITATION),
AND W. W. BUCKINGHAM, M.D.****
MT. VERNON, MO.

DARLING in 1906 gave the name *Histoplasma capsulatum* to round or oval bodies found in the endothelial cells and monocytes in certain cases of splenomegaly. This was found in autopsy material from patients in Panama.[1] In 1934, deMonbreun isolated the organism by culture from a clinical case and proved it to be a fungus.[2] Since 1934, much attention has been given to this disease, and we now identify the organism, *Histoplasma capsulatum,* as the sole etiological agent for this disease. Hodgson and associates[3] summarized 138 proved cases of histoplasmosis in 1950. In 65 of these cases there was reported to be pulmonary involvement. Forsee and co-workers[4] presented 30 cases of focalized pulmonary histoplasmosis following histopathologic examination. No recent tabulation of reported cases of histoplasmosis is available, but isolated reports of proved cases have markedly increased in recent years.

Various classifications of histoplasmosis have been presented. Conant, Smith, and others[5] present 3 distinct types: (1) primary histoplasmosis, (2) reinfection histoplasmosis, and (3) progressive histoplasmosis. Furcolow[6] is preparing to present a clinical classification based on symptomatology.

Since our series is composed primarily of pulmonary pathology, with only one focalized lesion, we will refer to the clinical form expressed by the term "chronic progressive pulmonary histoplasmosis" (as suggested by Furcolow). This clinical form could be classified under reinfection histoplasmosis, utilizing the types just mentioned. This type of histoplasmosis is of particular interest because of the similarity to chronic, progressive, cavitary tuberculosis, and we feel our experience substantiates this view.

Chronic progressive pulmonary histoplasmosis (reinfection type) was not assumed to exist until 1948 when Johnson and Batson[7] and Bunnell and Furcolow[8] presented original cases of pulmonary histoplasmosis with multiple

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From the Department of Surgery, Missouri State Sanatorium, Mt. Vernon, Mo.

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calcified areas in the lungs indicating healing. Sutliff and his associates[9] published cases similar to these and used the term "active chronic pulmonary histoplasmosis." Their patients presented calcifications in the lungs and cavity formations in the upper lobes, suggesting fibrocaseous tuberculosis. Sputa were negative for the tubercle bacillus, the tuberculin skin test varied, and the histoplasmin skin test was generally positive. Final proof in the diagnosis was established by finding the organism, *Histoplasma capsulatum*.

The Missouri State Sanatorium lies on the periphery of the endemic belt which includes the Mississippi and Ohio River Valleys and runs along the Appalachian Mountains. When numerous patients presented findings similar to those just described and pulmonary tuberculosis could not be proved, chronic progressive pulmonary histoplasmosis was suspected. Eighteen proved cases of pulmonary histoplasmosis have now been found at our institution. Seven patients have been subjected to surgery and 5 more were treated with MRD-112 (beta-diethylaminoethyl-fencholate).

MATERIAL

These 12 proved cases of chronic progressive pulmonary histoplasmosis form the basis of this report. Tabulation of some of the data as to the surgical and medical cases in this report shows a similarity to cases previously reported. Two of the 12 patients had pulmonary tuberculosis in addition to pulmonary histoplasmosis. Seven of the patients were submitted to surgery; 6 of these without drug therapy and 1 receiving MRD-112 for sixty days. The remaining 5 were treated with MRD-112. The ages ranged from 37 to 62. All were men except two; only one Negro patient was in the series. The duration of illness ranged from six to sixty-seven months. One patient gave a history of pulmonary disease for twenty-five years, but a diagnosis of histoplasmosis was not proved. The lesions were confined to the upper lung fields in 8 cases. In 4 cases there was bilateral pulmonary involvement. In 2 of these 4 cases, the disease had progressed to complete destruction of the left lung with apical infiltrations on the right. Eleven patients presented fibrocavitary disease and the other one presented nodular tuberculoma-like lesions.

All patients presented upper respiratory symptoms with the exception of the patient who presented nodular disease. This patient was completely asymptomatic. We considered the disease to be active because none of the patients could be employed at the time of admission to the hospital. All symptoms proved to be progressive; repeated x-rays revealed increased disease. Table I summarizes the surgical cases.

Five of the patients who survived surgery have returned to normal life. One patient died on the tenth postoperative day following a massive pulmonary embolus. One patient died sixty hours following left thoracotomy with acute pseudomembranous enteritis with toxic hepatitis and toxic nephrosis. The remaining 5 (to be reported later) are still h[illegible] ceiving aminostilbamidine or no drug therapy.

TABLE I. SURGICAL CASES OF HISTOPLASMOSIS
STATE SANATORIUM

NAME	AGE	ONSET	X-RAY FINDINGS	SKIN TEST		ORGANISM FOUND	LATEST HISTOPLASMIN SEROLOGY	LENGTH OF OBSERVATION (MONTHS)	TREATMENT AND COURSE
				TBC	HISTOPLASMIN				
S. R.	58	1/52	Multiple cavities, right lung	–	+	Histo + Sputum + Lung	C/F Test: 0 Prec. Test: 0	37	Right pneumonectomy; 18 mo. p.o. improved and working
E. M.	53	7/52	Cavity and infiltration, right lung	+	+	A.F.B. + Sputum Histo + Lung	C/F Test: 0 Prec. Test: 0	32	Dual segmental resection; 17 mo. p.o. improved and working
H. F.	41	10/53	Cavity and infiltration, RUL	–	+	Histo + Sputum + Lung	C/F Test: 0 Prec. Test: 0	18	Right upper lobectomy; 13 mo. improved and working
H. H.	37	9/53	2 inspissated cavities, left lung	–	+	A.F.B. + Lung Histo + Lung	C/F Test: 0 Prec. Test: 0	19	Wedge resection left side; 10 mo. p.o. improved and working
G. M.	46	12/53	Infiltration and cavity, RUL	+	–	Histo Lung	C/F Test: 8 Prec. Test: 40	6	Right upper lobectomy; 5/13/54, dead pulm. embolism—10 days
D. B.	43	10/53	Cavity, RUL	+	+	Histo Lung	C/F Test: 8 Prec. Test: 0	16	Right segmental resection; 8 mo. p.o. working
A. D. E.	56	12/48	Cavities, left lung; bilateral infiltration	+	+	Histo + Sputum + Lung	C/F Test: 32 Prec. Test: 40	67	MRD-112 experiment; left thoracotomy; dead

Histo = *Histoplasma capsulatum.*

REPORT ON CASES WITHOUT DRUG THERAPY

CASE 1.—S. R., a 59-year-old white woman, was first admitted to the Missouri State Sanatorium on Feb. 23, 1953 (Fig. 1). She stated that in January, 1952, she had had cough, hemoptysis, was easily fatigued, and had lost weight. The productive cough remained, weight loss was persistent, and the sputum remained streaked with blood on occasion. In January, 1953, she had symptoms consistent with cholecystitis. An x-ray of the chest revealed disease in the right lung, and she was advised by her physician to enter the Sanatorium. At the time of admission she had lost 20 pounds, had begun to have night sweats, and the cough and hemoptysis persisted. Mild dyspnea on exertion was noted.

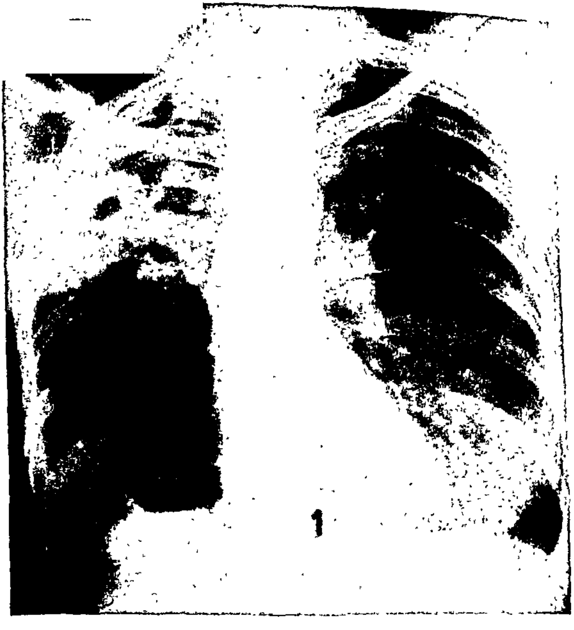

Fig. 1.—Case 1. A 59-year-old white woman with proved pulmonary histoplasmosis.

The family history and past history were noncontributory. Physical examination revealed a well-developed, chronically ill, white woman, showing signs of recent weight loss. Fine moist râles were heard over the right upper lung field. The blood pressure was 120/74. The remainder of the examination was not remarkable. Complete blood studies were normal. Repeated sputum examinations, including cultures, were negative for acid-fast organisms. Tuberculin skin tests, first and second strengths, were negative. Coccidioidin and blastomycin skin tests were also negative. Histoplasmin skin test was positive. The patient remained in the hospital as a diagnostic problem. Bronchoscopic examination, including smears for malignant cells and acid-fast organisms, was negative. On Sept. 23, 1953, a sputum culture was reported positive for *H. capsulatum*. Two days later, a right pneumonectomy was performed. The patient had a relatively uneventful postoperative course. Cultures from the resected lung were positive for *Histoplasma capsulatum* and the organism was also identified in the tissues. Serologic studies prior to surgery were stable at 1:16 on the complement fixation tests. Ten days postoperatively the complement fixation titer rose to 1:32 and one month following surgery was 1:64. Since that time the strength of the reaction to the complement fixation test has declined and at the present time is 1:8, as are also the precipitin tests for histoplasmosis. The patient has now been followed for eighteen months and is completely asymptomatic. She has gained 20 pounds in weight and has returned to her duties as a housewife.

CASE 2.—E. M., a 53-year-old Negro woman, developed a productive cough, fever, chills, and weight loss in September, 1952. These symptoms persisted, and in December, 1952, she developed pleurisy on the right side. She was admitted to the Missouri State Sanatorium on Feb. 27, 1953. X-ray on admission revealed infiltration in the right upper lobe and the superior segment of the lower lobe with definite cavities in these locations. Physical examination was noncontributory except for moist râles over the right lung. Urinalysis and hemograms were normal. Smears and cultures were positive for acid-fast organisms, and a diagnosis of pulmonary tuberculosis, far-advanced, active was made. The patient was started on isonicotinic acid, hydrazide (INH), 4 mg. per kilogram of body weight, and streptomycin, 1 Gm., twice weekly. A pneumoperitoneum was instituted on April 14, 1953, and was discontinued Feb. 5, 1954. The sputum converted after three months of drug therapy. The tuberculin skin test was positive, as was also the histoplasmin test. On Oct. 21, 1953, the posterior segment of the right upper lobe and the superior segment of the right lower lobe were resected. The postoperative course was uneventful. Acid-fast organisms were not found in the resected specimen, but *H. capsulatum* could be demonstrated in the lung tissue. The patient was discharged on April 1, 1954, with two diagnoses: (1) pulmonary tuberculosis, far-advanced, arrested, and (2) pulmonary histoplasmosis, inactive. Sputum cultures have remained negative for both organisms since surgery, the serologic titer is negative, and the patient is working.

CASE 3.—H. F., a 41-year-old white man, developed a productive cough, malaise, and pain in the right upper chest in August, 1953 (Fig. 2). A diagnosis of "flu" was made by his local physician but, after the symptoms continued, an x-ray of the chest was made in November, 1953, and the infiltration and cavitation in the right upper lobe were found. He had a weight loss of 20 pounds in four months. He was admitted to the Missouri State Sanatorium on Dec. 2, 1953. Physical examination revealed only moist râles in the upper right chest. Otherwise, the examination was noncontributory. Routine laboratory studies were normal. Twenty sputum smears and three cultures failed to reveal acid-fast organisms. Bronchoscopy on Dec. 21, 1953, was negative. Skin tests were as follows: tuberculin, 3 plus; histoplasmin, 3 plus; blastomycin, 1 plus; and coccidioidin, negative. Sputum cultures were reported positive for *Histoplasma capsulatum.* Serologic studies revealed a complement fixation titer of 1:8. On Feb. 10, 1954, a right upper lobectomy was performed. The patient had an uneventful postoperative course and was discharged on March 17, 1954. Repeated cultures since surgery have failed to reveal *H. capsulatum.* The serology is now negative for histoplasmin. The patient has returned to his usual occupation of farming.

CASE 4.—H. H., a 37-year-old white man, dates the onset of his illness to September, 1953, when he had a routine chest x-ray made, and was told he had a shadow in the left lung. He displayed no symptoms referable to the chest. He was admitted to the Missouri State Sanatorium on Feb. 6, 1954. Admission x-rays revealed a solid lesion in the left lung field in the mid-portion which was interpreted as a tuberculoma or a neoplasm. Physical examination was noncontributory. Urinalysis and hemograms were normal. Tuberculin skin test was negative, but the histoplasmin skin test was strongly positive.

Bronchoscopy was not remarkable. Repeated smears and cultures were negative for acid-fast organisms and fungi. The patient refused surgery until a second admission. On April 26, 1954, a wedge resection of two tuberculomalike lesions was performed. Both lesions were located in the lower lobe. The postoperative course was uneventful. Frozen sections revealed a granuloma similar to tuberculosis. Consequently, the patient received streptomycin and para-aminosalicylic acid (PAS) immediately after surgery. Cultures from the specimen were reported positive for *H. capsulatum* and acid-fast organisms. The Schubert histoplasmin complement fixation and the Salvin histoplasmin precipitin tests were negative prior to surgery and have remained so since surgery. The patient has returned to work but we plan to continue antituberculosis therapy for one year postoperatively.

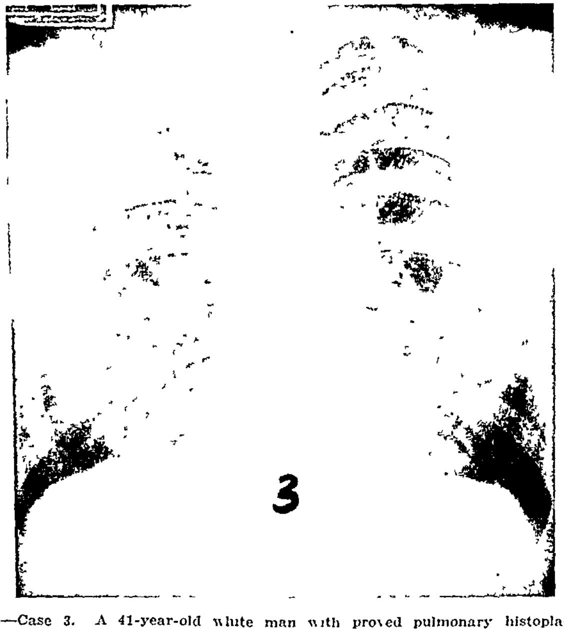

Fig. 2.—Case 3. A 41-year-old white man with proved pulmonary histoplasmosis

Case 5.—G. M., a 46-year old white man, had generalized aches, productive cough, and chills in December, 1953. A local physician treated him with penicillin, and after an x-ray of the lungs was made, he was admitted to the State Sanatorium on Jan. 4, 1954. X-rays of the lungs on admission revealed infiltration and cavitation in the right upper lobe. Physical examination revealed moist râles over the right apex, an amputated third finger on the right hand, and an inguinal hernia. Urinalysis and hemograms were normal. Skin tests were: tuberculin, 2 plus; and fungus skin test, negative. Twenty sputum examinations and three cultures failed to reveal acid-fast organisms. Sputum cultures for fungi were also negative. Complement fixation test for histoplasmosis was 1:8 and the precipitin test was 1:40 on May 7, 1954. On May 12, 1954, a right upper lobectomy was performed. *Histoplasma capsulatum* was easily identified in the lung tissue. The patient was ready for discharge on the tenth postoperative day, when he became cyanotic, markedly dyspneic, and complained of a terrific pressure in the chest. The blood pressure and pulse could not be obtained. He died two hours later in spite of heroic therapy. Autopsy revealed a massive embolism of the main pulmonary artery.

CASE 6.—D. B., a 43-year-old white man, had a cough with streaked sputum in October, 1953. The blood-streaked sputum continued intermittently until an x-ray was made in the Outpatient Department of the Sanatorium. This showed a cavity in the right upper lobe and a suspicious area of cavitation in the left upper lobe. He stated that he also had a pleuritic pain in the right chest on occasion. Following his admission to the Sanatorium on Jan. 19, 1954, routine urinalysis and hemograms were normal. The physical examination was noncontributory. Three cultures and numerous sputum smears were negative for acid-fast organisms. Repeated complement fixation titers for histoplasmin demonstrated a rising titer from 1:4 to 1:16 over a four-month period. Sputum cultures failed to reveal *Histoplasma capsulatum*, however. On June 7, 1954, an apical-posterior segmental resection was performed. His postoperative course was uneventful. Tissue cultures were reported positive for *H. capsulatum* and negative for acid-fast organisms. He was discharged on June 22, 1954, and has returned to his farm. Repeated cultures for fungi have been negative since surgery and the complement fixation titer is now 1:8. Segmental resection of the cavitary lesion on the left has been recommended.

CASES TREATED WITH MRD-112

Six additional patients were proved to have chronic progressive pulmonary histoplasmosis by culture of the organism from the sputum. The severity of this type of disease and the similarity to cavitary tuberculosis suggested that if drug therapy were beneficial, surgical resection for destroyed lung components would be safer. The spread factor in the surgery of histoplasmosis can be compared to the spread which occurred in tuberculosis before the use of antituberculosis drugs. There was experimental evidence that the antifungus agent, MRD-112,[10] would be beneficial in this respect. This drug is technically known as beta-diethylaminoethyl-fencholate.* A dosage of 150 mg. daily, intravenously, was selected and first administered to 5 patients starting on June 1, 1954.

On Aug. 1, 1954, the case next presented was selected for a left upper lobectomy. X-rays of Cases 7, 8, and 12, who received MRD-112, are presented (Figs. 3, 4, and 5).

CASE 7.—A. E., a 56-year-old white man, was first seen in the Outpatient Department of the Missouri State Sanatorium in December, 1948, after a mobile unit chest x-ray revealed pulmonary disease. He was followed in the Outpatient Department through July, 1953. All sputum examinations were consistently negative for acid-fast organisms. He had experienced progressive dyspnea since 1945. In August, 1953, after cutting silage, he experienced a cough, productive of purulent material, fever, and chills. He received streptomycin and penicillin from his local physician. The streptomycin was continued for two months. On Oct. 5, 1953, he was admitted to the Sanatorium, with a history of weakness, fatigue, and a weight loss of 15 pounds prior to admission. Physical examination was negative except for moist râles scattered over the left upper lung field posteriorly. The blood pressure was 130/84. The admission x-ray revealed bilateral infiltration in the apical portion of each lung. Several definite cavities were present in the left upper lung field. No definite cavities were seen on the right. All sputum examinations, including cultures, were negative for acid-fast bacilli. Bronchial smears were also negative for malignant cells and acid-fast organisms. Skin tests were: tuberculin, 3 plus; histoplasmin, 4 plus; blastomycin and coccidioidin, negative. Sputa were reported positive for *Histoplasma capsulatum* in December, 1953. Serum sent to Dr. Middlebrook for tuberculin complement fixation was negative. The patient had received INH and PAS from Oct. 23, 1953, to December, 1953, when the diagnosis of pulmonary histoplasmosis was established. He was discharged in December, 1953, to be followed in the Outpatient Clinic. Histoplasmin complement fixation was positive on April 1, 1954, at 1:128.

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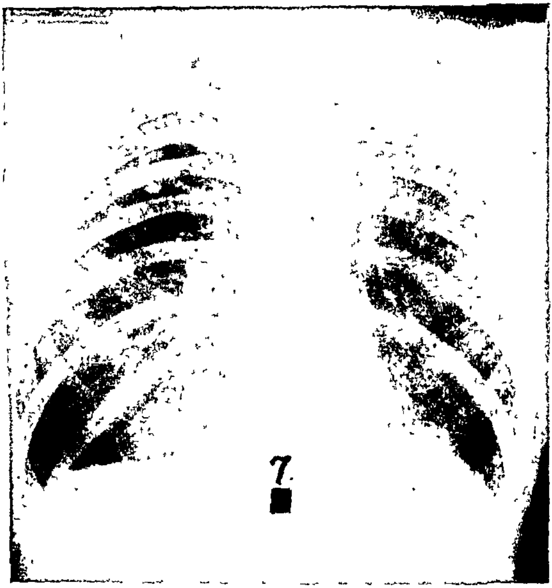

Fig 3.—Case 7. A 56-year-old white man with proved pulmonary histoplasmosis.

On May 26, 1954, the patient was readmitted to participate in the controlled histoplasmosis study (Fig. 3). *Histoplasma capsulatum* was again cultured from the sputum on two occasions. Smears and cultures were again negative for the tubercle bacillus. Routine laboratory work up and special liver and kidney tests were run. No evidence of liver or kidney damage was found, and the patient was started on MRD-112, 150 mg. intravenously daily, on June 1, 1954. Four other patients were started on this drug at the same time. After sixty days of therapy, with no obvious improvement, we advised a left upper lobectomy in an attempt to remove the cavitary components. On Aug. 13, 1954, a left exploratory thoracotomy was done. A large cavity with extensive reaction was found in the upper lobe; however, the superior segment of the lower lobe was markedly involved and the basal segments of the lower lobe were emphysematous and cystic. A pneumonectomy would have been necessary to remove the disease, and, with the infiltration in the right lung, we decided against this surgery.

lobectomy was performed. *Histoplasma capsulatum* was easily identified in the lung tissue. The patient was ready for discharge on the tenth postoperative day, when he became cyanotic, markedly dyspneic, and complained of a terrific pressure in the chest. The blood pressure and pulse could not be obtained. He died two hours later in spite of heroic therapy. Autopsy revealed a massive embolism of the main pulmonary artery.

CASE 6.—D. B., a 43-year-old white man, had a cough with streaked sputum in October, 1953. The blood-streaked sputum continued intermittently until an x-ray was made in the Outpatient Department of the Sanatorium. This showed a cavity in the right upper lobe and a suspicious area of cavitation in the left upper lobe. He stated that he also had a pleuritic pain in the right chest on occasion. Following his admission to the Sanatorium on Jan. 19, 1954, routine urinalysis and hemograms were normal. The physical examination was noncontributory. Three cultures and numerous sputum smears were negative for acid-fast organisms. Repeated complement fixation titers for histoplasmin demonstrated a rising titer from 1:4 to 1:16 over a four-month period. Sputum cultures failed to reveal *Histoplasma capsulatum*, however. On June 7, 1954, an apical-posterior segmental resection was performed. His postoperative course was uneventful. Tissue cultures were reported positive for *H. capsulatum* and negative for acid-fast organisms. He was discharged on June 22, 1954, and has returned to his farm. Repeated cultures for fungi have been negative since surgery and the complement fixation titer is now 1:8. Segmental resection of the cavitary lesion on the left has been recommended.

CASES TREATED WITH MRD-112

Six additional patients were proved to have chronic progressive pulmonary histoplasmosis by culture of the organism from the sputum. The severity of this type of disease and the similarity to cavitary tuberculosis suggested that if drug therapy were beneficial, surgical resection for destroyed lung components would be safer. The spread factor in the surgery of histoplasmosis can be compared to the spread which occurred in tuberculosis before the use of antituberculosis drugs. There was experimental evidence that the antifungus agent, MRD-112,[10] would be beneficial in this respect. This drug is technically known as beta-diethylaminoethyl-fencholate.* A dosage of 150 mg. daily, intravenously, was selected and first administered to 5 patients starting on June 1, 1954.

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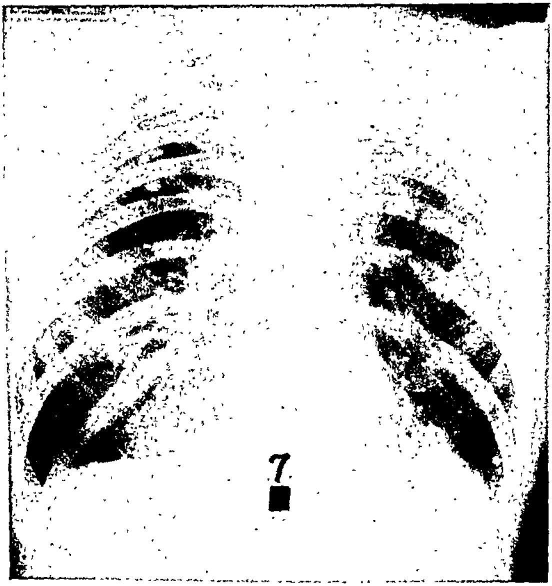

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Approximately four to six hours after surgery the patient showed signs of shock which did not respond to whole blood, Levophed, etc. Nine hours after surgery he vomited 3,000 c.c. of coffee-ground material. Drainage consisted of only 150 c.c. of blood into the thoracotomy bottles. He died in irreversible shock sixty hours postoperatively, after receiving 8 whole blood transfusions, with anuria for twenty-four hours. Permission for autopsy was obtained.

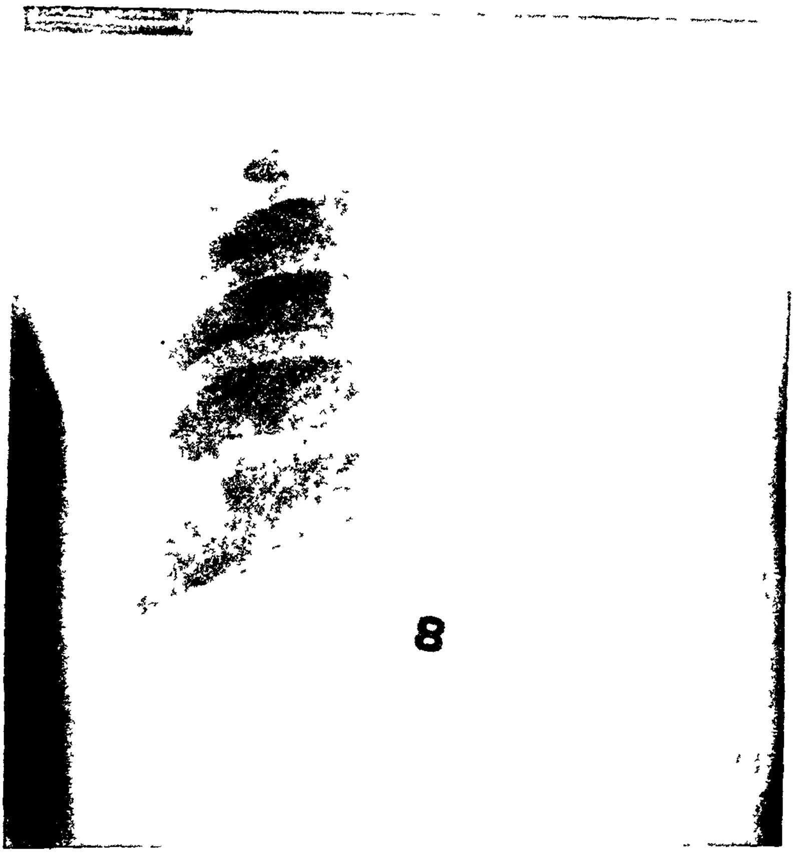

Fig. 4.—Case 8. A 67-year-old white man treated medically with MRD-112

The pertinent findings from autopsy were: The parietal pleura on the left was markedly thickened and fibrous and there was an inflammatory reaction between the left lung and the chest wall. The middle segments of the left lower lobe presented a thin-walled emphysematous bulla. Throughout the lower lobe were numbers of discrete firm nodules. The superior half of the upper lobe and the superior segment of the lower lobe presented a large irregular density over which the thick pleura was noted. Beneath this pleura was revealed, on section, a thin-walled irregular-shaped cavity, measuring 3 cm. in longitudinal dimension. The inner wall was granular and reddish in appearance. The tissue around this was nodular, presenting numerous tiny cavities and, at the lower portion of the cavity, there was a communicating bronchus. The right lung showed numerous discrete, hard nodules which measured up to 8 mm. and were found to be present in the lower and middle lobes. There was marked infiltration throughout the upper lobe.

Gastrointestinal Tract: The stomach was somewhat dilated and contained approximately 250 c.c. of grayish brown material. The mucosa lay free. The first portion of the duodenum contained bile-stained mucoid material. The jejunum and ileum revealed a red, friable mucosa which could be easily scraped away. No discrete ulcerations were present. The cecum, ascending, transverse and descending colons were filled with dark, tarry stool which had the consistency of soft putty. The mucosa was intact and pale red in appearance.

Microscopic: Lungs: Sections from the area of the grossly described cavitary lesion revealed an irregularly outlined wall, the inner aspect covered with acidophilic-staining necrotic material. This overlay a zone of fibrotic reaction. The cells showed a tendency to picket-fence formation. Subjacent to this there was a loose fibrovascular area in which multinucleated foreign body giant cells and inflammatory cells were seen. Several bronchi showed disfigurement of the walls with desquamation of the epithelium. Tissue stained with acid-fast bacilli (AFB) and PAS stains failed to reveal the presence of acid-fast organisms or of intracellular inclusion bodies. Cultures taken from the cavity were positive for *H. capsulatum*.

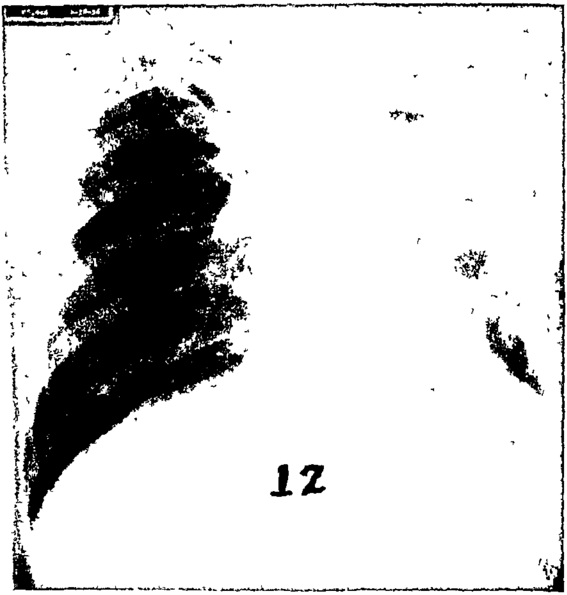

Fig 5—Case 12 A 61-year-old white man treated medically with MRD-112.

Liver: Sections showed extensive central and mid-zone parenchymal necrosis The cells showed loss of nuclei, marked granularity, and swelling of the cytoplasm. The peripheral zones showed swollen liver cords and hyperchromatic nuclei.

Kidneys: Sections of the cortex showed very extensive degeneration of both the proximal and distal tubules. The glomerular tufts were moderately swollen and showed some amorphous deposit. Within some of the tubules was seen accumulation of slaty gray deposit. The medullary portion presented moderate interstitial edema and the tubular epithelium appeared intact. The histologic features were those of nephrosis involving the corticotubular system.

Small intestine: Sections showed pronounced loss of granular epithelial mucosa. In some areas the villous processes had sloughed off and instead there was a hemorrhagic edematous inflammatory tissue. In others, the villous processes presented marked hemorrhagic extravasation. Throughout the wall there was much edema and numbers of eosinophils and neutrophils. The histologic feature was that of subacute enteritis.

Final Pathologic Diagnosis: (1) Acute pseudomembranous enteritis with toxic hepatitis and toxic nephrosis complicating irreversible postsurgical shock, and (2) pulmonary histoplasmosis, bilateral.

Following this tragedy, no further surgery on these patients was contemplated until long-term observation and study could be carried out. These cases will be published in detail later, but several observations concerning the drug, MRD-112, can be made at this time. First, no benefit has been observed in any patient with chronic progressive pulmonary histoplasmosis treated with MRD-112. One patient treated with this drug suffered a spread to the opposite lung three months after therapy. Two have shown increased symptoms of cough, hemoptysis, and weight loss. These two have also shown a marked rise in the serologic titer. All continue to have sputa from which *H. capsulatum* can be cultured, and 4 of the 5 remaining cases show varying degrees of liver degeneration, as proved by liver biopsy.

PATHOLOGY OF RESECTED AND AUTOPSY SPECIMENS

Puckett[11] reviewed the pathology in resected cases and was able to identify the organism, *Histoplasma capsulatum,* in 22 cases. Four well-defined groups were found, varying from a localized pneumonitis to a cavity with satellite tubercles. A survey of the pathologic findings in our series revealed only 1 case in which the lesion resembled an isolated nodule similar to a tuberculoma. In the remaining cases, all revealed cavities of varying size, from 2 to 5 cm. in diameter. Numerous smaller cavities were scattered around the periphery. The cavities contained caseonecrotic material and were lined by smooth friable walls. The inflammatory reaction extended to the pleura, creating a thickened fibrotic pleura which could not be separated from the underlying lung. An extrapleural resection was required in 6 cases. The pleura in Case 1 measured 1½ cm. in thickness.

On microscopic examination the cavities were found to be filled with cellular detritus and caseonecrotic material. The areas of necrosis were bordered by a zone of fibrocytic reaction containing fibroblasts, plasma cells, mononuclear macrophages, and foreign body giant cells. Single and conglomerate tubercle-like formations were observed and, in 4 of the cases, irregular-shaped refractile bodies were found within the giant cells. The bronchi within and adjacent to the cavities showed varying degrees of bronchiolitis.

Periodic acid-Schiff stains were a valuable aid in the demonstration of the fungi in tissues of 5 of our cases. Peabody and co-workers[12] have recently reported on the value of this stain in demonstrating fungi in tissues of pulmonary granulomas. These authors suggest that since fungi cannot be cultured from such small nodular lesions in many cases, this might represent the healing phase of such disease Furcolow,[13] in 1948, was of the opinion that organisms in calcified lesions might not be viable We certainly would agree with this, but if a breakdown occurred, then a spread of the disease might occur similar to the spread which occurs after the sloughing of a tuberculous inspissated cavity

SEROLOGIC STUDIES

Serologic studies have been carried out in detail on all patients by the U S Public Health Service, Kansas City, Kan, through the cooperation of M L Furcolow, M D, Medical Director All the resulting titers cannot be tabulated, but in Table II examples of the manner in which we have tried to utilize this data are shown Until more experience is gained in the knowledge of serologic studies, caution in their interpretation must be used, but we feel certain facts have been gained

TABLE II SEROLOGY RESULTS

CASE 1 (S R)			CASE 9 (W D)			CASE 7 (D E)		
	HISTOPLASMIN			HISTOPLASMIN			HISTOPLASMIN	
DATE	CF*	PPT†	DATE	CF	PPT	DATE	CF	PPT
8/ 1/53	32	80	12/17/53	neg	80	5/ 1/54	1 64	40
8/13/53	1 32	80	4/ 5/54	1 16	1 40	5/28/54	1 256	40
9/25/53	Operation		6/ 1/54	MRD 112		6/ 1/54	MRD 112	
9/28/53	16	80	6/11/54	1 8		6/ 5/54	1 32	40
10/ 2/53	16	80	10/22/54	32	100	6/22/54	1 32	40
10/ 6/53	32	80	12/17/54	1 32	100	7/23/54	1 16	
10/24/53	64	80	2/ 8/55		80	8/ 7/54	1 32	
5/21/54	8	40				8/13/54	Operation—died	
9/ 1/55	8	8						

The serology on S R is negative The serology on W D has continued to rise since MRD 112 was administered We feel this precludes surgery The findings at surgery on D E revealed extreme activity This was Case 7

*Complement fixation

†Precipitin

We feel that the highest titers represent the highest degree of activity of the disease Cases are considered positive and active when the titer is higher than 1 4 on either the complement fixation test or 1 10 on the precipitin test It is from such patients that the organism has been isolated A significant titer, using the yeast phase antigen, denotes early disease, whereas, chronic disease is suggested by a significant titer using the broth filtrate antigen, histoplasmin In an attempt to apply this knowledge as an aid in operative intervention, we now wait until a titer of 1 16 or less is obtained Case 7 had a complement fixation titer of 1 32 and a precipitin titer of 1 40 Marked activity was demonstrated at autopsy, with positive cultures being obtained from the cavity in the left lung and from a hilar node

Complete serologic studies have been utilized in an attempt to follow the postoperative cases Four of the 5 have negative titers and 1 has a complement fixation level of 1 8 Should any case show a rise in the serologic titers, medical therapy in the form of MRD 112 or aminostilbamidine would be instituted, because we feel this would represent relapse or reactivation of the histoplasmosis

DISCUSSION

The effects of medical therapy have been very disappointing in all forms of histoplasmosis. Christie and colleagues[14] reported that 5 of 12 patients with the disseminated form of the disease had recovered following treatment with ethyl vanillate. Other forms of therapy have included sulfadiazine, bacillomycin, Aureomycin, streptomycin, stilbamidine, isoniazid, and actidione. Chronic progressive pulmonary histoplasmosis, in spite of its insidious course, has never responded to any form of therapy either. Michael and Vogel[15] reported on the use of MRD-112 in the treatment of a disseminated case of histoplasmosis. Six cases of chronic progressive pulmonary histoplasmosis treated at the Missouri State Sanatorium with MRD-112 showed no response to this drug.

Pulmonary resection, as reported by several others,[16, 17] appears to offer progress in the therapy of pulmonary histoplasmosis. With the exception of coccidioidal disease, it has not been advocated widely for other pulmonary mycotic infections. Our experience confirms this concept and suggests several indications for resection.

Sutliff, Hughes, and their associates[9] stated as an indication for surgery: a localized lesion without evidence of tuberculosis. Pulmonary coccidioidomycosis and tuberculosis occurring as coexisting diseases do not seem to influence each other adversely.[18, 19] Our limited experience with tuberculosis and histoplasmosis suggests that these diseases do not affect each other alarmingly. We feel that if histoplasmosis and tuberculosis are localized together, and that if adequate antituberculosis therapy has been administered, resection is definitely indicated. We feel that a person weakened by chronic progressive pulmonary histoplasmosis and remaining in a tuberculosis sanatorium, with or without a diagnosis of tuberculosis, should receive antituberculosis therapy prior to resection.

The only spread to the contralateral lung in the 12 patients mentioned in this report occurred from a large cavity in the right upper lobe containing fluid. We had recommended resection, but the patient refused surgery. This bronchiogenic spread might have been avoided if the patient had accepted resection. We prefer to resect all cavities containing fluid in patients who have pulmonary histoplasmosis. In our opinion, this indicated resectional surgery.

Localized or circumscribed pulmonary lesions which remain undiagnosed warrant early thoracotomy. A period of six weeks for study and diagnosis seems warranted in selected cases. One of our cases in whom cancer was suspected fitted into this category, and both acid-fast organisms and *Histoplasma capsulatum* were found in the cultured tissues.

Even though 2 of the patients in this series had segmental resections and 1 had wedge resection, it is now our belief that lobectomy would be the operation of choice. Further follow-up studies and operative experiences will be needed before this final observation can be completely ascertained.

The role of collapse therapy in histoplasmosis, thoracoplasty or plombage, has never been studied thoroughly. In cases of chronic progressive pulmonary histoplasmosis, thoracoplasty prior to resection may be indicated in an attempt to stabilize the disease. We do not feel that thoracoplasty or plombage alone would have any beneficial effect on the type of disease which we have been observing at this hospital.

Last, if a drug becomes available which will protect the patient from a spread of the histoplasmosis or from a flare-up of stable disease, resection for destroyed areas of the lung would appear to be justified.

Follow-up examinations have been performed routinely on all resected patients. In all of the patients who survived surgery, the complement fixation studies have either remained stable or become negative. A rise in the serologic titer would indicate reactivation of the disease and medical therapy, such as MRD-112 or aminostilbamidine, would be given.

CONCLUSIONS

1. Medical therapy in chronic progressive pulmonary histoplasmosis has been disappointing. Six patients treated with MRD-112, a new antifungus agent, have shown no response to therapy.

2. Pulmonary resection appears to offer some progress in the therapy of chronic progressive pulmonary histoplasmosis. It has not been widely advocated and further experiences will be needed before final evaluation as to this form of therapy can be ascertained.

3. Pulmonary histoplasmosis and tuberculosis occurring as coexisting diseases are not uncommon. Antituberculosis therapy prior to resection, if the diseases are localized together, appears justified.

4. Serologic studies have proved to be of benefit in the preoperative preparations and postoperative follow-up of surgical cases. It is suggested that resection be withheld until the disease is stabilized as judged by the complement fixation test or until the strength of the reaction is 1:8 or less. Further observation with relation to the complement fixation studies is also necessary.

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DISCUSSION

DR. J. W. PEABODY, JR., Washington, D. C.—As a guest of the Association, I would first like to express my gratitude for the privilege of the floor. Having incorrectly inferred from the title of this paper that we might be at odds about the merits of MRD-112 in treating histoplasmosis, I should apologize for rising to discuss Dr. Polk's splendid presentation, for it is apparent that we are all agreed on the relative ineffectiveness of this compound. Actually, our entire experience with the chemotherapy of progressive histoplasmosis has been most discouraging. Because of previous disappointments with any number of drugs exhibiting in vitro effectiveness superior to that of MRD-112, we were naturally somewhat skeptical prior to its use. The following case confirmed our doubts and is worth recording, since it provides the best possible evidence of its clinical inefficacy.

(slide) This is the admission chest roentgenogram of a 40-year-old white male patient from Dr. J. D. Murphy's service at Oteen, N. C., showing bilateral exudative disease with left apical cavitation. Sputum was repeatedly negative for acid-fast bacilli, but bronchial washings grew out *Histoplasma capsulatum* as did many subsequent sputum specimens.

(slide) Here following an interval of bed rest, note the general improvement although the cavitary disease in the left apex seems better defined. This patient then received 150 mg. of MRD-112 daily, for twenty-three days, only to have a follow-up roentgenogram (slide) disclose an extensive spread to the left lower lobe. Without further drug therapy the latter density subsequently cleared. He has since had a thoracoplasty and will probably require a lobectomy.

All of this simply illustrates that as yet we have not found a satisfactory therapeutic agent, unless of course the toxicity of nystatin can ultimately be circumvented.

While a great many other points might be mentioned, two in particular deserve emphasis, namely, the prevalence of histoplasmosis and its remarkable similarity to both primary and reinfection types of tuberculosis. It is fast becoming evident that regardless of one's geographic location, histoplasmosis will be recognized proportionately to one's interest and suspicion. For example, we now have resected over 50 pulmonary granulomas in more than one-third of which *Histoplasma capsulatum* can be demonstrated by special stains. (slide) This photomicrograph shows nothing more than caseation necrosis, a finding once considered prima facie evidence of tuberculosis. The same tissue section, however, after decolorization and restaining by the periodic acid-Schiff method (slide) discloses myriads of *Histoplasma capsulatum.* This is not to imply that the method is new; it is simply incredible that it should be so slow in being accepted. Most pathologists still need to be coaxed into giving the PAS and Gridley stains a decent trial.

Finally, coexisting histoplasmosis and tuberculosis should not be overlooked, a situation we have encountered at least three times. (slide) This patient presented a cavitary lesion in the left apex. Sputum was positive for acid-fast bacilli. Three cavities were found in the resected specimen, but only one of these contained tubercle bacilli. The other two cavities, including the one visualized so nicely in this planigram, contained *Histoplasma capsulatum* in tissue sections and grew out *Histoplasma capsulatum.* Such findings emphasize the need for very careful evaluation of every granulomatous process.

The authors are to be commended for a very timely and stimulating presentation.

DR. FELIX HUGHES, Memphis, Tenn.—I hope if you have any lingering doubts that histoplasmosis is a separate entity, and an entirely different disease, that this paper and the discussion will help to dispel it. With a patient who appears to have tuberculosis but has never had a positive sputum, the probable diagnosis of histoplasmosis should be made. The figures presented in San Francisco from the Fitzsimons group were on resection of granulomas caused by histoplasmosis with little active disease. The prognosis is good at the onset of the disease, and the patient may not even be sick. Then it may give rise to multiple calcifications, and I think histoplasmosis is the cause of many of the calcifications that we have in the past attributed to pulmonary tuberculosis. I know you have seen the cavities that have been present with histoplasmosis in Dr. Polk's slides and Dr. Peabody's slides.

(slide) Here is another that was removed before the diagnosis was made. We recently had a patient with all the symptoms of an acute lung abscess, who was treated with all the antibiotics, and acute cavitary disease was found when the resected lesion was opened. The microscopic picture was that of caseous tuberculosis. However, instead of culturing tubercle bacilli, only the *Histoplasma capsulatum* grew on the culture media. At VAMTG Hospital (Kennedy Hospital) we have had 16 cases and we do not believe that MRD-112 is of great benefit. For cavitary lesions of histoplasmosis the complications of resections may be compared to resection of active open tuberculous cavitary lesions with bacilli resistant to chemotherapeutic agents as shown by Dr. Welles and his associates, in the first paper presented this morning. Dr. Murphy has 1 patient he thinks was cured by thoracoplasty, and some type of collapse procedure may be of benefit in treating patients with bilateral cavitary lesions.

I agree with Dr. Sutliff that the time appears appropriate to survey the patients under treatment for pulmonary tuberculosis to determine the incidence of pulmonary histoplasmosis both as a separate disease and as a complicating lesion. Routine culture for fungi of all resected pulmonary lesions is also recommended.

DR. SHELDON E. DOMM, Knoxville, Tenn.—East Tennessee has a high incidence of histoplasmosis, and the observation of cavitary lesions in our practice is not uncommon. One case undergoing resection may be worthy of a brief report.

The patient (G. E. H.) was a 45-year-old white man. X-ray showed a 2 cm. cavity in the left upper lobe. Tuberculin skin test was negative. Histoplasmin skin test was strongly positive. The sputum was negative for acid-fast bacilli but was positive for *Histoplasma capsulatum.*

The patient underwent segmental resection of the apical-posterior segments of the left upper lobe by Dr. W. K. Rogers. Pure culture of *H. capsulatum* was obtained from scrapings of the cavity lining. (Slide.) The pathologic report was histoplasmosis. The patient enjoyed an uneventful postoperative course, and follow-up seven months postoperatively shows no recurrence or complication. The patient did not receive MRD-112 or other specific chemotherapy.

We have recently observed bilateral thin-walled histoplasmosis cavities of three years' standing in a woman patient who had previously undergone right middle lobe lobectomy for bronchiectasis. Bilateral resection is contemplated.

We would like to stress the danger of overgrowth by *H. capsulatum* following indiscriminate antibiotic therapy in any patient from the endemic area of histoplasmosis. Certainly the routine use of prophylactic antibiotics postoperatively in cases of resection for histoplasmosis may be inadvisable.

DR. BUCKINGHAM (Closing).—In a diseased condition which so closely resembles chronic, fibrotic, cavitary pulmonary tuberculosis roentgenologically, the question has been asked how we suspect the diagnosis. In all hospital admittances, routine tuberculin and Histoplasma skin tests are done. If the histoplasma skin test is positive then further investigation is made from the serologic standpoint. This disease is not limited, or at least the types we have seen, by the normal anatomic barriers. It extends through the pleura into the intercostal spaces through the segmental planes and through the interpulmonary fissures, and it has been our thought that under these circumstances lobectomy certainly should be the procedure of choice. We have seen 1 case of the so-called solitary granulomatous lesion which the pathologist reported as granulomatous reaction; the acid-Schiff stain is almost specific in demonstrating the fungi in the tissues. We agree with the other discussants that the drug has not been of any great help.

When we first presented this paper for acceptance we thought we might have something of value in MRD-112, but as we got into the work more and more we realized that the drug was not of any specific help in this condition. Liver biopsies in all the cases treated with MRD-112 showed evidence of damage.

Dr. Crafoord asked what MRD is; it is an experimental drug which the Merrell Company has been working on. It has done a great deal of experimental work on fungus drugs.

THE DEVELOPMENT OF THE PLEURAL PARTITION TO PREVENT OVEREXPANSION OF THE LUNG FOLLOWING PARTIAL PULMONARY RESECTION

AN EXPERIMENTAL STUDY WITH CLINICAL APPLICATION

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INTRODUCTION

EXTENSIVE resection of one or more lobes, or multiple segments of the lung results in the creation of a large intrapleural dead space. If this space is not adequately eliminated at the time of operation, overexpansion of the lung leading to lung rupture, increase in the postoperative complications, prolonged postoperative morbidity, and finally decreased pulmonary function may result. The usual methods of controlling the space have certain inherent disadvantages. In searching for a more physiologic method, the concept of fashioning an intrapleural partition was evolved as a means of effectively preventing overexpansion of the remaining pulmonary tissue on the operative side. Experience with the production of 36 pleural partitions of various materials in mongrel dogs is presented along with the clinical application of this procedure.

EFFECT OF A LARGE INTRAPLEURAL DEAD SPACE ON REMAINING PULMONARY TISSUE

There is no question that if only a small pleural dead space is produced by the lobectomy the remaining lung tissue will expand to fill this cavity. It is especially true in children, where the alveoli apparently have not only the ability to distend but actually to *increase in number*. In adults, however, the lung generally expands by means of dilatation of the alveoli. This may progress to the formation of generalized emphysema or emphysematous bullae *which so increase the residual air as to decrease greatly the pulmonary function* of the unresected lung tissue. If significant amounts of pulmonary fibrosis are present in the remaining pulmonary tissue, dilatation of the alveoli is hampered and the lung expands but little. On the other hand, if considerable emphysema is present in the remaining lobe, then the expansion of this lobe to fill the pleural space results in the formation of large emphysematous blebs or bullae which further decrease the pulmonary function. In some instances, actual rupture of these emphysematous blebs may take place.

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If the residual lung does not expand to fill a large pleural dead space, immediately after operation, certain other mechanisms come into play. The diaphragm rises and usually is fixed. The mediastinum shifts to the affected side, provided firm mediastinal adhesions are not present. The chest wall contracts by narrowing of the intercostal spaces. Finally, pleural fluid is formed, much as the so-called "fluid ex vacuo" in artificial pneumothorax, when a nonexpansile lung is present. These mechanisms all tend to decrease the pulmonary function on the operative side. However, of these mechanisms, the formation of large amounts of pleural fluid and the raised and fixed diaphragm most seriously depress the respiration of the remaining pulmonary tissue. In order to decrease the size of the pleural dead space after lobectomy, various operative procedures have been employed.

COMMON METHODS OF DECREASING PLEURAL DEAD SPACE

For over half a century a number of surgical procedures have been carried out to provide collapse of the lung in pulmonary tuberculosis. With the introduction of the antimicrobial drugs to destroy the tubercle bacilli and with the great technical improvements that have come with pulmonary resection, these collapse measures are now being used less and less as primary procedures. However, these collapse measures are still employed to decrease the pleural dead space following partial pulmonary resection. Diaphragmatic paralysis is the simplest and probably the most common method of decreasing this residual space. Once the diaphragm is relieved of its downward contraction, it will readily rise because of the positive intra-abdominal pressure and the negative intrapleural pressure, to obliterate even moderate intrapleural dead spaces. If the space is large, however, then this usually is insufficient and it may be necessary to add pneumoperitoneum. The latter alone is also done without actual paralysis of the diaphragm. This is effectual in obliterating small pleural defects. High levels of the diaphragm, however, cannot be immediately effected without phrenic paralysis or a previous extensive pneumoperitoneum. If the diaphragm is maintained for a long time at a high level in the thoracic cavity it becomes stretched and thin. Most of the contractility of the muscle fibers is then lost. The weak and thinned-out diaphragm cannot contribute greatly to the dynamics of respiration and there is a corresponding decrease in pulmonary function on the operative side. Thus, the simplest means of controlling the dead space in the pleural cavity may result in permanent loss of diaphragmatic action which may greatly decrease the pulmonary function.

The performance of thoracoplasty to collapse the chest wall has long been a common means of controlling the pleural dead space following lobectomy. This is an effectual method of closing this cavity and has the advantage of not impairing diaphragmatic function on the operated side. It has been used for many years throughout the civilized world and probably is the most generally accepted method of managing large pleural defects, especially in pulmonary tuberculosis. The mortality is low and it is a highly successful operation. However, the drawbacks are well known. First, is the deformity of the thoracic cage as well as the prolonged pain and discomfort [illegible] pany this operation. Of course, the more limited [illegible] plasty, the less deformity and disability. However, if [illegible] moved, then the shoulder disability and pain may [illegible] the muscle imbalance and pain may severely decrease the [illegible] of the operated side, even though the diaphragm has [illegible] tainly all thoracic surgeons, who have grown up in the [illegible] have had an appreciation of the discomfort, deformity, and [illegible] function which the patient suffered in order to be well, [illegible] a simple, effectual and safe technique to take its place.

Since the beginning of this century, surgeons have inserted [illegible] body prostheses into the extrapleural and pleural planes, to [illegible] pulmonary collapse for active pulmonary tuberculosis. More [illegible] technique has been used to decrease the pleural dead space following [illegible] monary resection. Although a host of substances have been tried, there [illegible] a few which apparently are quite well tolerated by the body. Among [illegible] paraffin, methyl acrylate (Lucite), zirconium, and Ivalon sponges. The [illegible] vantages of these foreign bodies are that they are light in weight, can be [illegible] inserted, and do not produce a great deal of foreign body reaction. The [illegible] advantages which are well known are those inherent with the introduction of foreign material in any amount into a body cavity or an artificially produced space. The use of foreign body plombage as a treatment of pulmonary tuberculosis has given way to the safer thoracoplasty and pulmonary resections in most clinics throughout the world. Following World War II, Lucite sphere plombage, introduced by Wilson and Baker,[15] gained some popularity as a prosthesis material to provide collapse in pulmonary tuberculosis. However, after considerable experience, the nonspecific and tuberculous infections that occurred in the spaces about the Lucite spheres have made surgeons hesitant to employ these spheres as permanent prostheses, although we believe this form of treatment still has its advocates. The writings of Trent,[11] DesForges and associates,[8] Wada,[12] and others emphasize the dangers of this material when used as a prosthesis. Plastic sponge (Ivalon) first used by Grindlay and co-workers[10] has been further reported by Gale and associates.[9] The latter workers have reported satisfactory results in 46 out of 50 cases; however, Brantigan,[2] Davis,[7] and Charbon and Adams[5] have reported a number of infections. We have used the Ivalon sponge as a prosthesis in the extrapleural plane in dogs. Because of the infections that were so produced we have not employed it in humans. Since the report by Gale and co-workers,[9] in 1952, we have been unable to come across a similar series of cases. Thus, experience with this type of sponge has not been extensive enough for one to have a final opinion regarding its use at this time. It may be, with improvements in sterilization and in the management of the space, that this, or some other foreign body, may be well tolerated. However, as Clagett[6] has stated in his comments on Gale's paper, there has been considerable disappointment with other foreign bodies used in human tissues in the past, nevertheless further trial of this method was justified.

THE DEVELOPMENT OF THE PLEURAL PARTITION

We have not been completely satisfied with the techniques of thoracoplasty, phrenic operations, and pneumoperitoneum, and the filling of the extrapleural or pleural spaces with considerable amounts of foreign body material. For some time we have been searching for a simple surgical procedure that would be safe and effectual and that could be carried out immediately following the lobectomy and yet preserve the respiratory function of the remaining lung tissue on the operative side. It occurred to one of us (L. A. B.) that it might be possible to divide the pleural cavity into two compartments: One containing the remaining lung, which could then function in a normal manner, freed of the disadvantages of overexpansion, extensive collapse of the chest wall, and the paralyzed attenuated diaphragm. The second compartment, isolated by the partition, would be nonfunctional and would gradually fill with fluid and fibrin clot. By fashioning the partition across the upper portion of the pleural cavity a nonfunctional apical space would be created whereas the excursion of the diaphragm and lower ribs would not be impaired.

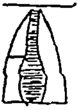
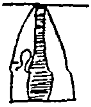
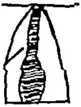
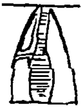
Therefore, to test out this concept of limiting pleural expansion, a series of 36 partitions were fashioned of various substances in mongrel dogs and the dogs were sacrificed at periods from one week to five and one-half months (Table I). The technique employed is the following: Under intratracheal ether anesthesia, lobectomy of one or more lobes was performed by the individual ligation technique. The bronchial stump was covered with a pedicled pericardial fat graft, according to techniques that we have previously reported both for experimental animals and humans.[3, 4] The pleural partition was then fashioned out of various substances and inserted into the pleural cavity.

TABLE I. TYPES OF PLEURAL PARTITIONS IN EXPERIMENTAL ANIMALS

PARTITION	NUMBER	DATE OF POSTMORTEM EXAMINATION
Tantalum mesh	7	35 to 148 days
Steel mesh	8	48 to 300 days
Nylon mesh	6	20 to 176 days
Fascia lata	15	7 to 180 days
Total	36	7 to 300 days

First, woven mesh of tantalum and stainless steel wire was used. Then about one year later, when on the basis of our clinical experience we decided that wire mesh should not be used, we started to employ nylon and then autogenous fascia lata to fashion the partitions. Following the removal of the lobe or lobes of the lung, the remaining lung tissue was inflated to maximum expansion by positive pressure applied through the intratracheal tube. The partition was then sutured in place at the level of maximum pulmonary expansion to divide the pleural cavity into two compartments. This partition was usually fixed at the level of the fourth to sixth rib. In the case of the partition formed with the woven wire mesh, the screen was fixed to the chest wall by interrupted silk sutures leaving a space from 2.5 to 3 cm. along the mediastinum to prevent trauma to the heart and great vessels from the mediastinal displacement occurring with respiratory and cardial movements. Either pleura or a pedicled pericardial fat graft was used to bridge the defect. In the case of the partitions formed with the woven nylon and the fascia, these partitions were fixed to the mediastinal tissues and/or pericardium, as well as to the chest wall. Following the fixation of the pleural partition, the chest wall was closed in layers with interrupted cotton sutures. Temporary closed catheter drainage of the inferior pleural compartment was maintained during closure to expel all the air present and expand the remaining pulmonary tissue. The catheter was then withdrawn. All dogs survived the surgery with the exception of 1 animal which died before the experiment could be completed, apparently from faulty action of the anesthetic machine. The dogs were then examined periodically to observe the fate of the partition and the secondary effects on the intrathoracic organs.

Table II Fate of Pleural Partition in Experimental Animals

Type of Partition	No.	Complete 	Incomplete 	Displaced Upward 	Pleural Membrane 
Tantalum mesh	7	3	4	7	7
Steel mesh	8	3	5	8	8
Nylon mesh	6	5	1	0	6
Fascia lata	15	15	0	0	15
Total	36	28	10	15	36

FATE OF THE INTRAPLEURAL PARTITION

What happened to the intrapleural partition is summarized in Table II. Chest roentgenograms of the dogs with various types of partitions are shown in Figs. 1 to 4. It was noted that the wire screen partitions were complete in 9 out of 15 instances, probably because no firm fixation was made between the wire screen partition and the mediastinum. Furthermore, the wire screens tended to be displaced upward. The lung herniated up a little into the superior compartment in those cases where the partition was incomplete. The woven cloth partitions, however, were complete in all but 1 case. There was little tendency toward the upward displacement and no contraction or pull on the mediastinum was noted. In one instance the [illegible] tially free in the superior space.

The autogenous fascia lata partitions were complete in all instances. There was no upward displacement. The partition was firm and strong with some pull on the mediastinum. The partition did show signs of contraction after the first month which was as much as from 20 to 50 per cent in three or four months. In no instance did the fascia partition pull free from the chest wall or mediastinum. There was no breakdown of the fascia so it formed an effectual barrier to prevent pulmonary expansion. All partitions were covered with a glistening membrane regardless of type of material used to form the partition.

Fig. 1.

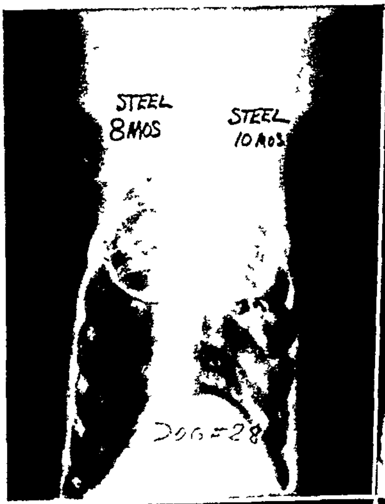
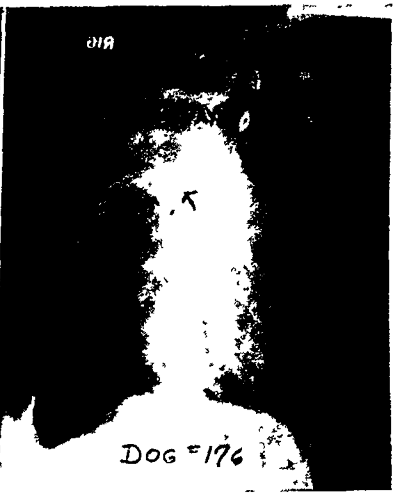


Fig. 2.



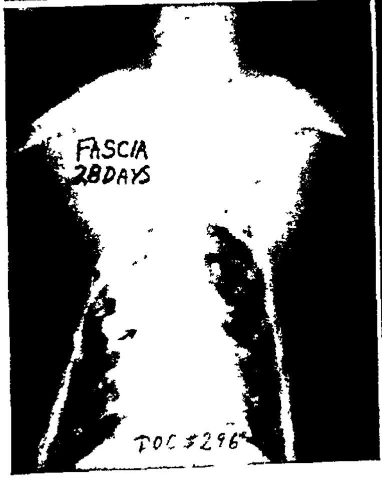


Fig. 3.

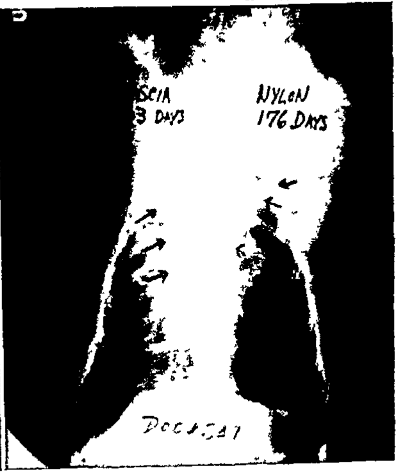


Fig. 4.

Fig. 1.—Dog 285. Chest roentgenogram of bilateral steel mesh partitions (right side) eight months and (left side) ten months after upper and middle lobe lobectomies. The lower pleural compartments are clear. Upper compartments cloudy. Partial herniation of lung on right through incomplete partition.

Fig. 2.—Dog. 176. Chest film with nylon partition 118 days after right upper and middle lobe lobectomy. Right lower pleural compartment clear. Slight mediastinal shift toward upper compartment which is cloudy. Partition complete.

Fig. 3.—Dog 296. Chest x-ray fascia lata partition 28 days after upper and middle lobectomy. Clear lower pleural compartment. Upper compartment hazy. No mediastinal shift. Partition complete.

Fig. 4.—Dog 247. Chest film of bilateral upper and middle lobe lobectomy with right fascia lata partition 63 days after surgery and left nylon partition 176 days after operation. Lower pleural compartments are clear. Upper compartments cloudy.

MICROSCOPIC STUDIES OF THE FASCIA PARTITIONS

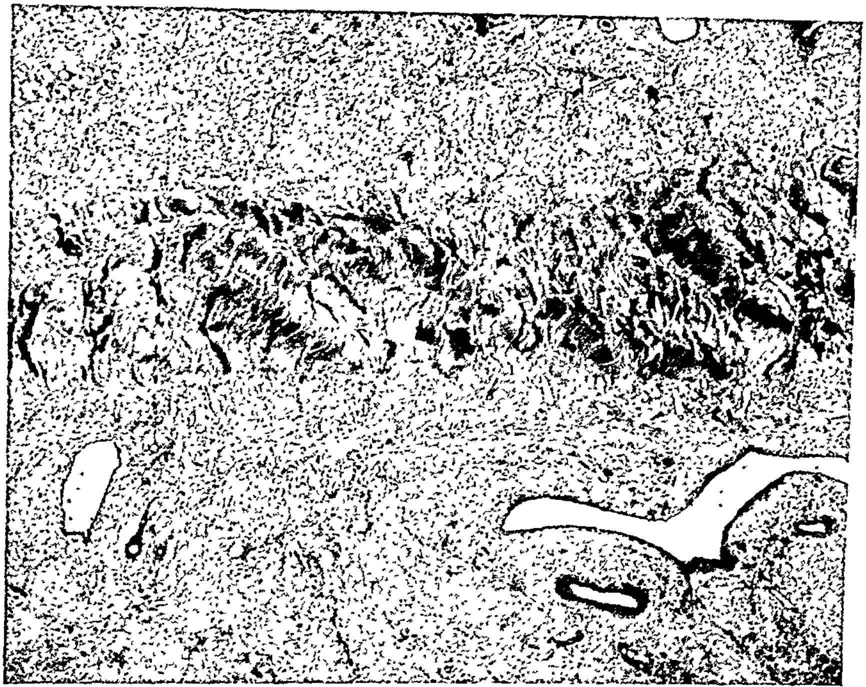
At the beginning of our use of fascia lata to form the partition in dogs there was considerable speculation as to the fate of these rather large grafts. Smaller grafts had been used for years in hernias and plastic operations and larger ones for chest wall defects.[15] Therefore, microscopic studies were carried out on these partitions beginning one week after the transplant to the pleural cavity up to five and one-half months. In one week's time (Fig. 5) the fascia lata was well preserved in the center of the partition between two layers of organized granulation tissue, two to three times the thickness of the fascia, containing an active fibroblastic proliferation with numerous capillaries and vascular spaces. There was a mild inflammatory reaction as evidenced by a sparse small round cell and neutrophil infiltration and a fibrinous exudate on the surface. Two weeks after transplant (Fig. 6) the granulation tissue was more densely fibrous although the fibrocollagenous fascia lata was well preserved in some areas. In others, replacement of the fascia with this fibrous granulation tissue had taken place. Inflammatory reaction was minimal.

In six weeks' time (Fig. 7), the original fibrocollagenous fascia was demonstrable as a narrow band in the center of the partition. This was integrated with the granulation tissue, which was now of dense fibrous and fibrocollagenous character. There were numerous capillaries and small blood vessels supplying the partition. These apparently arise from the parietal pleura and the adherent lung. The fascia lata fibers made up only about one fourth of the thickness of the partition which was composed of adult connective tissue with only a sparse infiltration with small round cells. After two months' interval (Fig. 8) the partition was entirely composed of a dense fibrocollagenous connective tissue of mature cells. It was impossible to distinguish between the original fibrous tissue of the fascia lata and that of the newly formed replacement fibrous tissue. It was a tough living tissue that had developed its own blood supply. Observation up to five and one-half months' time have shown no change in the microscopic findings just described for the two-month sections. Therefore, these studies reveal that the fascia lata remains viable for a period of about six weeks' time after which it becomes completely replaced with an adult fibrocollagenous connective tissue which, having its own blood supply, is a living fibrous partition. In one instance where the partition was formed with nylon mesh, the nylon had separated from the chest wall and was lying partially cured up in the superior space. However, a tough fibrinous membrane separated the pleural compartments.

OBSERVATIONS ON THE SUPERIOR PLEURAL COMPARTMENT

In animals with all types of partitions there was a slight shift of the mediastinum to the resected side and the intercostal spaces over the superior pleural compartments were somewhat contracted. Though the fragile mediastinal pleura was found to be tented and displaced to the operated side we do not believe there was any functional impairment of the structures of the mediastinum. Observations were made up to six mon [illegible] regardless

Fig. 5.



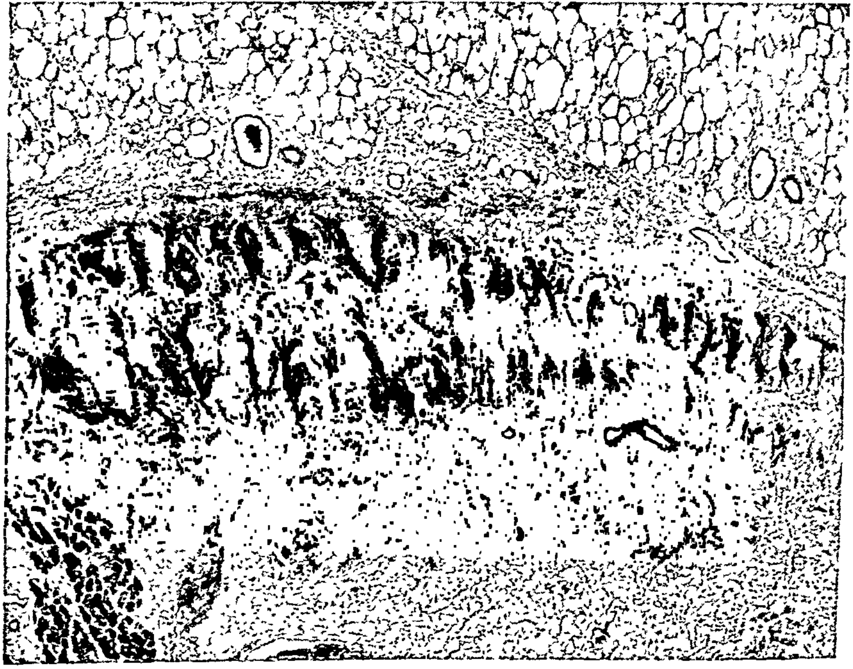
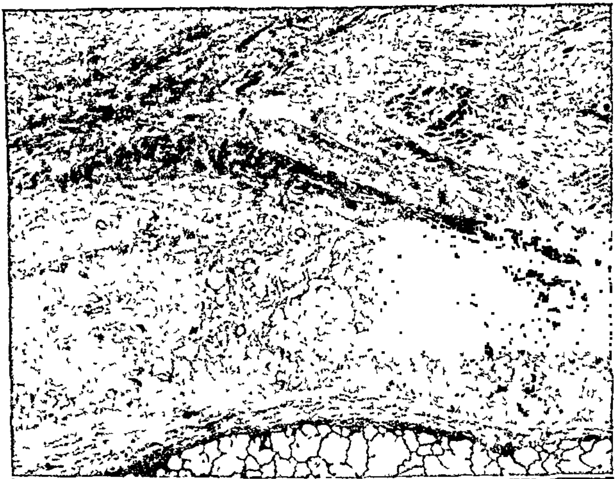


Fig. 6.

Fig. 5.—Dog 299. Fasca lata partition one week after transplantation. Fibers of fascia well preserved covered on each side by an organized granulation tissue made up of an active fibroblast proliferation with numerous vascular spaces and capillaries. Mild inflammatory reaction present.

Fig. 6.—Dog 264. Fascia lata partition two weeks after transplantation. Fascia viable, but is being replaced by a densely fibrous granulation tissue with numerous capillaries. Adipose tissue clinging to graft along superior border and a small bundle of muscle fiber in lower left-hand corner are well preserved.

Fig 7



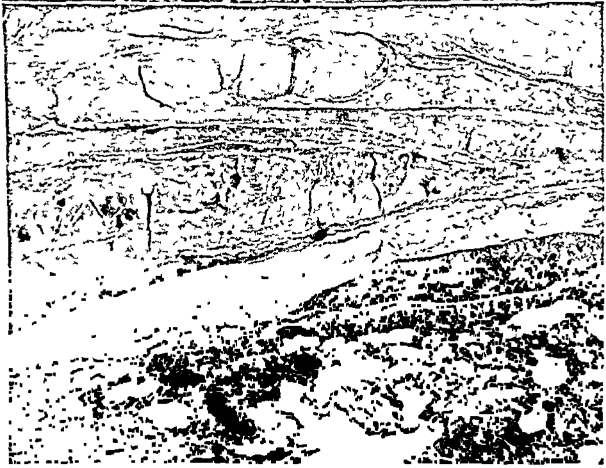


Fig 8.

Fig 7 —Dog 299 Fascia lata partition six weeks after transplantation Fascia appears as a narrow band of fibrocollagenous tissue in center of partition. Adult connective tissue replacing fascia makes up most of partition Fat cells and sparse muscle bundles still present Thickened pleura and lung seen along inferior border of partition

Fig 8 —Dog 266 Fascia lata partition two months after transplantation Partition now composed of mature fibrocollagenous connective tissue Newly formed connective tissue cannot be told from original fascia layer Scarred pulmonary tissue where lobar fissure had been incomplete is seen along inferior border.

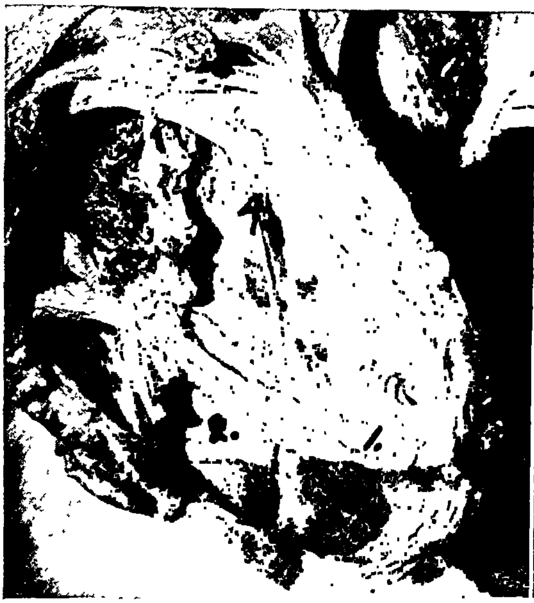


Fig. 9.

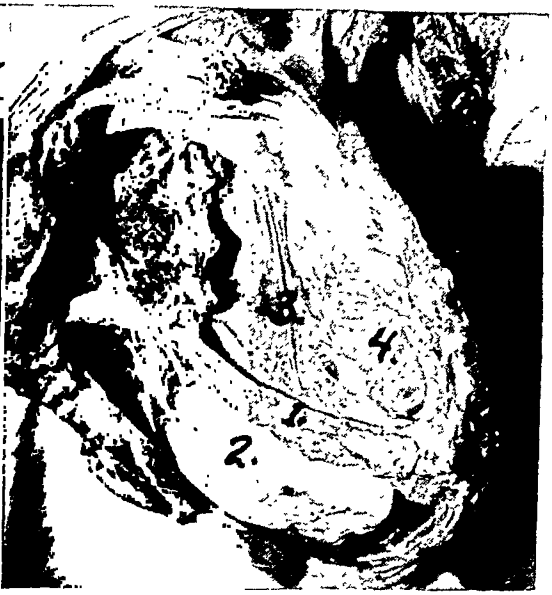


Fig. 10.

Fig. 9.—Dog 295. Twenty-two days after right upper and middle lobectomy with fascia lata partition. View, right lateral thorax, bony cage removed. Upper and lower compartments seen from above. Fascial partition (*1*) intact. In lower compartment collapsed lower lobe (*2*) resting on smooth diaphragm (*3*), which is normal. Upper compartment divided by phrenic nerve (*4*).

Fig. 10.—Dog 295. View of right lateral thorax same as Fig. 9, with right lower lobe expanded with positive pressure on intratracheal tube. Note how right lower lobe (*2*) expands normally to fill lower pleural compartment below partition (*1*). Phrenic nerve (*3*) and anterior mediastinum (4) visible in superior compartment.

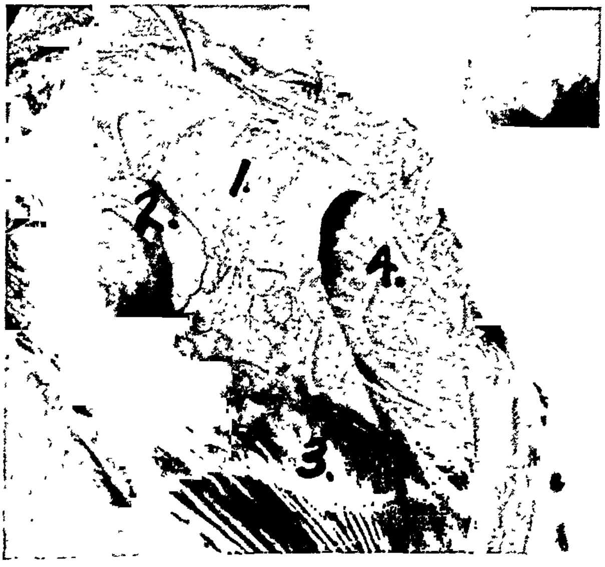


Fig. 11.—Dog 295. View of inferior pleural compartment showing fascial partition seen from below (*1*), collapsed lung free in the inferior pleural space (*2*), glistening diaphragm (*3*), and anterior mediastinal pleura (*4*).

of the material used to form the partition the superior space was completely lined with a shiny membrane in all instances where the pleura was complete.

EFFECT ON THE PLEURA AND LUNG IN THE INFERIOR INTRAPLEURAL COMPARTMENT

The most gratifying observation made in these experiments was the condition of the pleura and the diaphragm in the inferior pleural compartment (Figs. 9, 10, and 11). Except for adherence of the superior aspect of the lung to the pleural partition, there was complete absence of any pleural adhesions in the inferior space. Most important was the freedom of the diaphragm from any adhesion or clot whatsoever. This permitted the diaphragm to move in a normal manner. Parietal pleura and chest wall showed neither fixation nor scar, so that maximal chest wall movement apparently was also preserved. This meant that, so far as the remaining lobes of the lung were concerned, maximum expansion and compression of this pulmonary tissue was preserved. This preservation of pulmonary function was borne out by the clinical observation of the dogs which showed no lack of activity, even though in about one half of the cases they had received bilateral lobectomy with only the lower lobes on each side remaining for the function of respiration. As far as the remaining pulmonary tissue was concerned, there was no evidence of pulmonary tissue was concerned, there was no evidence of pulmonary emphysema. Microscopic examination showed the alveoli to be normal and similar to that of normal alveoli before surgery. These findings are most significant, and point to the fact that the intrapleural partition had achieved the purpose for which it was intended, with preservation of pulmonary function in the remaining lung.

CLINICAL APPLICATION

Although this paper concerns itself mainly with our experimental investigation that has been going on for a year and one half, the early results in human beings should be mentioned. To date our clinical experience is limited to the use of this procedure on only 9 cases (Table III). With the success of the wire mesh in chest wall replacement in humans and armed with our experience in dogs, we began to make a clinical trial of this technique using tantalum mesh about one year ago. Our first 5 cases did very well and we thought that this was the answer to the problem. In the next 2 patients, who were exceedingly bad risks (Cases 6 and 7), a secondary infection developed in the superior pleural compartment. With the occurrence of the secondary infection, we stopped employing the pleural partition operation clinically and turned to the laboratory again to find new materials out of which to form the partitions. We believed it was the material out of which we were fashioning the partition and not the principle that was at fault. After six months' experimenting with nylon and autogenous fascia in experimental animals we became convinced that of all the materials used, autogenous fascia lata was the most satisfactory one to form the partition. We were encouraged by the fact that the large fascia lata grafts used by Watson and James[14] to close chest wall defects had proved successful an apparently were well tolerated. Our application of the fascial graft relied on blood supply coming in from the pleura of the chest wall, and mediastinum; in their use of the fascia new blood vessels developed from the overlying muscles.

TABLE III. CLINICAL EXPERIENCE

CASE	DIAGNOSIS	OPERATION	PARTITION	DIAPHRAGM	INFECTIONS		MONTHS	RESULT
					SUP.	INF.		
E. C. 43 M	TB	R U L	T. M.*	Ex.	No	No	12	Working
B. N. 39 F	TB	R U L	T. M.	Ex.	No	No	12	Working
H. W. 39 M	TB	R U L	T. M.	Ex.	No	No	3	Died; coronary
B. B. 29 F	Bronchiectasis	L L L Lingula	T. M.	Ex.	No	No	10	Working
O. H. 40 F	Cyst	L L L	T. M.	Ex.	No	No	7	Working
R. S. 70 M	CA	L L L	T. M.	Ex.	+	0	7	Space sterilized; working
C. C. 37 F	TB	R U L R M L, and D L L	T. M.	Ex.	+	0	10	Apical cavity; working
D. B. 55 M	TB	R U L D L L	Nylon	Ex.	0	0	First day	Died; pulmonary embolus
K. H. 55 M	TB	R U L S M L S L L	Fascia	Ex.	0	0	1	Early OK

*T.M. = Tantalum mesh.

And, so during the past month we have again started employing the partition operation in human beings, using fascia lata with success in one case. The early results were excellent but long-time observations are not available. It will be noted for the entire series that the results were excellent in all patients surviving surgery with the exception of 1 case. In 2 cases in which the partition was formed out of wire mesh, a secondary infection had developed in the superior pleural compartment. It was of interest that in both cases the lung completely filled the inferior pleural space and the pleura, lung, and diaphragm were not involved. Furthermore, a limiting membrane rapidly formed to encase both surfaces of the wire screen which completely isolated the infection in the superior compartment from that of the inferior space. Both these patients were extremely poor risks and we believe they would not have tolerated either thoracoplasty or phrenic crush with an extensive pneumoperitoneum to fill a huge dead space. In both cases the infection was drained several weeks after surgery, when the patient's condition had stabilized. In one instance it was possible to sterilize the superior pleural space with antibiotics so that the drainage tube could be removed. The patient now has a solidly healed chest wall and there is no evidence of infection in the superior space. He is well and working as a cab driver. In the second case, in which we classify the result as only fair, the cavity is slowly decreasing in size. It is now about 30 c.c. However, the patient's pulmonary condition was so poor that, in spite of the infection in the superior pleural space, we believe that the preservation of the vital basal segments of the lower lobe of the right lung saved the patient's life, for the contralateral lung was

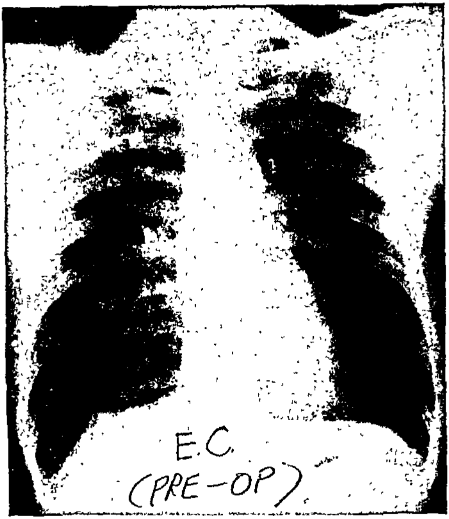


Fig. 12.—Case 1, E. C. Preoperative chest film. Pulmonary tuberculosis, right upper lobe. Bilateral pulmonary emphysema.

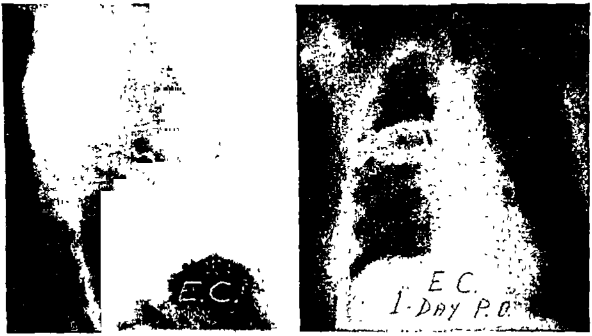


Fig. 13.—Case 1, E. C. Posteroanterior chest film one day after operation. Right upper lobe lobectomy with tantalum mesh partition to fourth rib anterior and fifth rib posterior. Lower pleural compartment, right, is clear. No fluid in upper compartment.

Lateral view shows position of tantalum mesh partition in relation to expanded middle and lower lobes. Note fissure between middle and lower lobes.

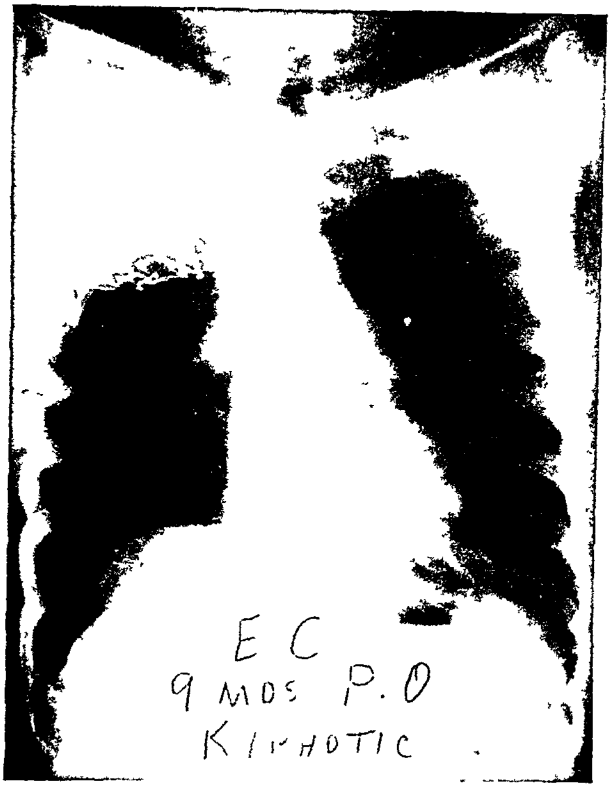


Fig. 14.—Case 1, E. C. Special kyphotic posteroanterior chest film months after operation Note central bulging of partition and clear diaphragmatic angles

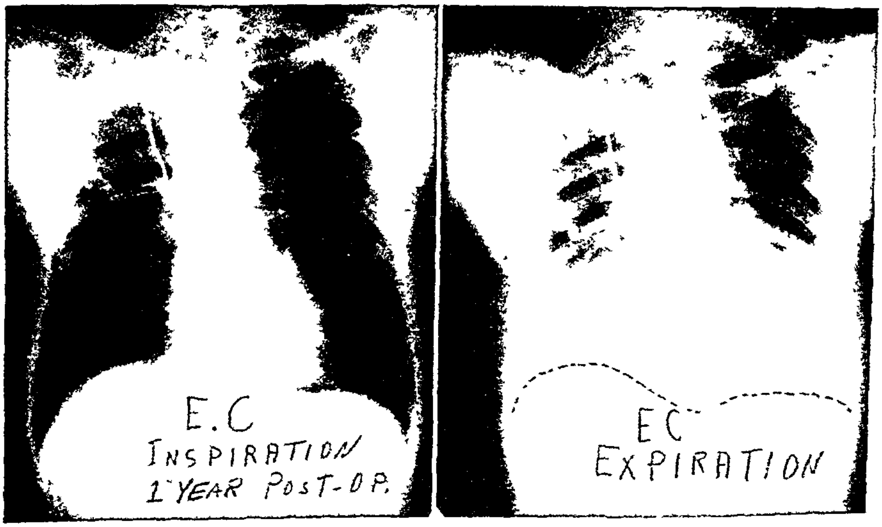


Fig. 15.—Case 1, E. C. One year after operation. Posteroanterior view on inspiration shows diaphragm at eleventh rib On expiration the diaphragm rises two and one-half inches Diaphragm action marked; contour completely free of adhesions or pleural reaction

practically functionless from fibrosis, permanent diaphragmatic fixation, and marked postpneumothorax pleural thickening. She desperately needs the remaining portion of the right lower lobe of the lung to carry out respiration. We believe the patient would not have survived an extensive thoracoplasty, or paralysis of the right diaphragm following the multiple lobectomies on the right. Because of the steady clinical improvement and decrease in the pleural space, we have delayed in the removal of the superior wire mesh screen and the performance of a limited thoracoplasty, which she now can tolerate from the point of view of pulmonary function.

The excellent results of pulmonary function in the other cases, especially Case 1, with pulmonary tuberculosis (Figs. 12, 13, 14, and 15) and Case 5, with an extensive emphysematous cyst of the lungs cannot be overlooked. In Case 5, no other form of surgical procedure which is commonly used today to decrease the pleural space would have been satisfactory. The vital capacity is increased from around 1,100 to 2,200 following surgery, and the patient is no longer bedridden and is now doing her own housework.

DISCUSSION

As far as the experimental evidence goes in the 36 partitions made in dogs, the results were excellent without any infection. This fact alone commends this procedure for further clinical trial. The pleural partition prevented overexpansion of the lung with the preservation of function of the diaphragm and chest wall movement on the operated side. Thus, we believe that this procedure is more physiologic than diaphragmatic paralysis with or without pneumoperitoneum or thoracoplasty. Wade[13] has recently made an elaborate study of the role of the thoracic cage and diaphragm in respiration. His investigation showed that the diaphragm contributed 75 per cent of the ventilation of the lung while the chest wall contributes the remaining 25 per cent. The diaphragmatic movement in quiet respiration is 1 to 2 cm. but may increase to 7 to 13 cm. with deep breathing. He showed the chest circumference in quiet respiration to increase from 1 to 2 cm. while with severe effort it may enlarge from 5 to 11 cm. For six to eight months after birth, respiration is almost entirely diaphragmatic, from which time on the chest begins to contribute to the ventilation of the lung. Although Best and Taylor[1] believe that diaphragmatic motion contributes only 60 per cent to ventilation as compared to the 75 per cent reported by Wade,[13] the preservation of the function of this structure is of tremendous importance to the poor risk patient.

The physiologic function of the apex of the lung is much less than that of the middle and lower portions, due to the lack of motion of the apices with respiration. Therefore, we believe that it is important to make the functionless compartment at the apex. In lower lobe lobectomy, by freeing up the hilus of the upper lobe of the lung, it is possible to form an apical compartment without undue torsion or poor drainage from the displaced upper lobe bronchus. Thus the ventilatory effect of the diaphragm and lower chest wall on the lung is preserved, for the upper lobe can then function like a lower lobe. This was done successfully on 3 dogs and in 3 of

The drawback of the partition operation is the possibility of infection in the superior space. Whether this will prove to be greater in the series of patients with fascia lata partitions than is found in simple lobectomy remains to be seen. From our limited experience we believe it would be higher in a large series with wire mesh partitions. The fascia lata has the advantage of becoming completely replaced by a living fibrocollagenous connective tissue with its own blood supply from the pleura of the chest wall and mediastinum. If an infection develops in this space above a fascia partition, simple drainage with or without a subsequent limited apical thoracoplasty, should solve the problem. Experience has shown that wire mesh usually must be removed in the presence of infection, and we plan to do this eventually in Case 7. The 2 cases in which there was a pleural infection differed from the ordinary postoperative generalized empyema in that the infection was limited to the superior compartment and the lower compartment was completely filled with lung tissue and remained uninfected. Thus the diaphragm and chest wall action was preserved even though an apical empyema developed. Furthermore, a late, limited thoracoplasty, we believe, can be tolerated by these extremely poor risk patients when the function of the remaining lung has returned to a near normal level.

SUMMARY

1. The concept of the formation of the pleural partition to prevent overexpansion of the lung has been presented.

2. Experience with the formation of 36 such partitions in dogs is recorded.

3. The operation was shown to be successful in the experimental animals.

4. Fascia lata was proved to be superior to tantalum, steel mesh, and nylon cloth as a material to form a partition.

5. The advantages of this procedure lie in the prevention of the overinflation of the remaining lung without sacrificing diaphragmatic and lower chest wall function.

6. The disadvantage lies in the possibility of localized infection of the superior compartment. However, this localized infection may not be as disabling as a generalized postoperative empyema.

7. Only limited application has been made (9 cases) in patients with huge pleural dead space, following lobectomy.

8. Early clinical results would indicate that further trial of this procedure is justified using fascia lata to form the partition. This trial should be restricted to those cases with a large intrapleural dead space.

We should like to express our appreciation to Florence Rhudy, M.D., for her assistance in the pathologic studies on the pleural partitions and to Lester Gibson, M.Sc., of the Hunterian Laboratory for technical assistance with the animal experiments.

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Discussion

DR. JOHN W. BELL, Sunmount, N. Y.—I think Dr. Brewer should be complimented on this fascinating contribution to the problem of intrathoracic space following resection surgery for tuberculosis. The use of a partition may be very helpful when such a space exists, particularly in the problem where the parietal pleura has been destroyed in mobilizing the upper lobe.

We have had experience with 25 pleural tents of the type described this morning. Our technique is similar to that described by Miscall and Klopstok and shown to you in their excellent movie. The pleural tents, or caps, have been fashioned at the time of operation at the conclusion of the resection. They have been very convenient in avoiding temporary space problems where they could be anticipated. I should like to make several technical points about these tents. Of course the pleural cap depends on the integrity of the parietal pleura. Where the pleura has been damaged by mobilization of the upper lobe it may be necessary to use a partition of some of the prosthetic materials selected by Dr. Brewer. All of our tents have been made from the parietal pleura. Occasionally there is a small rent in the pleura and air will leak up from the intrapleural space, even though it is drained with two tubes, into the extrapleural space above the tent. This may produce a tension pneumothorax which will dissect into the tissues and give alarming subcutaneous emphysema. This possible accumulation of air must be watched for and it may be necessary to decompress the extrapleural pocket with a third tube. The tent has been very useful in our hands in avoiding transient space problems.

No evidence has been presented today or in the literature that I know of, that tents, or even thoracoplasties for that matter, prevent overexpansion. Disregarding the fact that overexpansion has, we think, little to do with reactivation of residual tuberculosis, the overexpansion that the surgeon is trying to prevent may, of course, already have occurred when the surgeon enters the chest. Contracted segments co-exist with hyperinflated segments or lobes, so that nature has anticipated the surgeon in many instances.

We think these tents or partitions should have wider application in avoiding space problems in resections for tuberculosis.

DR. EDGAR DAVIS, Washington, D. C.—I rise for two comments only, first, [illegible]arding the use of Ivalon sponges and, se[illegible] tent.'' As you may recall, several y

Ivalon sponges, I issued a word of warning about the hazards of this prosthesis. Inasmuch as it continues to be used, I would like to report that we have now completely abandoned its use. Almost all our patients in whom it was used have got into trouble, some as long as three and four years afterward.

Second, a number of years ago we started to do a procedure called an extrapleural apicolysis for the purpose of preventing overexpansion of the partially resected lung. It is interesting to see the same procedure now proposed as a new idea under the descriptive term "pleural tent." I feel that the bad results obtained from extrapleural apicolysis were due to the fact that a plombage was introduced at the time of surgery. Following lobectomies we soon learned that if we merely pulled the pleura down as was described this morning and sutured it in much the same manner as the present authors describe, we could obviate complications by deferring the introduction of a plastic prosthesis for a week or ten days. Then a small incision could be made in the apex, the fluid and fibrinous exudate removed and the space filled with Lucite spheres. So far as I recall we have used this in some 60 patients and have so far had no reason to regret it.

DR. BREWER (Closing).—I wish to thank the discussants for their remarks. Their expression of the dangers of the use of large amounts of foreign body material in the extrapleural and intrapleural planes certainly reflects an experience that is widespread today. We have tried to use parietal pleura to form the partition. Pleura is usually fragile and will not hold the suture so that this material has been unsatisfactory in our hands. A thickened or inflamed pleura once freed from its blood supply might be the source of infection. The problem of drainage is exceedingly important. We employ two drainage tubes both anteriorly and posteriorly and can see where three might be advisable. One must be careful to place one of the drainage tubes at the extreme apex of the inferior pleural compartment whether they be placed anteriorly or posteriorly. In some instances two should be placed at the apex of the pleural compartment if there is leakage of air from an emphysematous lung. The drainage of fluid has not offered a serious problem. This is probably true because the lung completely fills the inferior pleural compartment to seal off this space, provided the air has been adequately removed by suction. In some of the early cases we did have air passing up into the superior pleural compartment with the result that subcutaneous emphysema occurred. The proper placement of the drainage tubes and constant negative suction will prevent this.

The dangers of use of tantalum mesh is well taken. As pointed out in the paper, we have abandoned the use of this material to form a partition, and now use fascia lata. In the 2 patients with a superior compartment in which tantalum mesh was used, there was no general pleural infection. The inferior compartment was free of infection and the function of the diaphragm, lower chest wall, and remaining lung was preserved. This was lifesaving in the woman who was depending upon the basal segments of the right lower lobe for respiration with a poor functioning contralateral lung. We doubt if she would have survived a generalized pleural infection.

However, infection is the only drawback that has been observed to date in this method, and the operator must be careful with the preoperative preparation of the patient, the operative technique, and the postoperative care, to prevent such infection. From a physiologic point of view this method to our knowledge preserves the pulmonary function on the side of the resection better than any other procedure currently used. At the present time we are undertaking studies to demonstrate just how successful this preservation of pulmonary function is. The advantage of fascia lata lies in the fact that it is not a foreign body, that it is completely absorbed and a living membrane takes its place. Even though one might encounter an apical infection in the superior compartment, simple drainage would probably take care of it in most instances, with a limited apical thoracoplasty the maximum that would have to be done. In several cases in which we have used the pleural partition, where thoracoplasty or phrenic paralysis of the huge pneumoperitoneum were contraindicated, we wondered what we would have done had not the technique of the pleural partition been available.

OPERATIVE THORACIC DUCTOGRAPHY

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ALBANY, N. Y.

INTRODUCTION

SUBSEQUENT to the first effective intrathoracic ligation of the thoracic duct by Lampson[9] in 1946 to control chylothorax, an increasing number of similarly successful surgical experiences have been reported.[1, 2, 3, 5, 11, 13, 16, 17, 20] Since the primary concern of the present article is not with the historical aspects of chylothorax nor its etiology, diagnosis, mortality, or experimental studies, the reader is referred to the many excellent investigations and reviews which have appeared in the literature.[2, 4, 8, 9, 10, 13, 18, 19] The purpose of this communication is to present the results of anatomic studies of the thoracic duct system as well as to record our experiences with a radiopaque technique for visualization of the thoracic duct at the time of its deliberate surgical interruption in the treatment of chylous effusion.

Demonstration of the thoracic duct by contrast roentgenography is neither a new nor an original concept. According to Scannell,[15] it was suggested by Churchill and successfully accomplished by their group in a dog with excellent delineation of the thoracic duct. Emerson in 1947 excised a supradiaphragmatic thoracic duct cyst and at operation injected the cyst with Diodrast, obtained a portable film, and proved communication of the cyst with the thoracic duct cephalad to it. Caudad injection was not performed. Anatomic studies of the thoracic duct in fresh post mortem subjects by such radiopaque techniques have also been reported.[6] Touroff and Seley[20] instilled Lipiodol into the mediastinum following excision of a mediastinal hygroma for chronic chylothorax. Apparently *no attempt was made by them to cannulate and visualize the duct,* the Lipiodol being employed to promote an inflammatory response and subsequent fibrosis of any residual lymphatic radicles not ligated. These observations suggested to us that routine radiopaque visualization of the thoracic duct system at the time of operation might prove a valuable adjunct to the surgical interruption of the thoracic duct by exposing accessory ducts to ensure their recognition and subsequent identification and division at the diaphragm. No reports advocating such use of the technique herein described have been found.

MATERIALS AND METHODS

In thirty instances, the thoracic duct has been studied by proximal and distal injection of radiopaque dye at the level of the diaphragm. The dye was

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From the Department of Surgery, Albany Medical College, Albany, N. Y.

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 24 to 26, 1955.

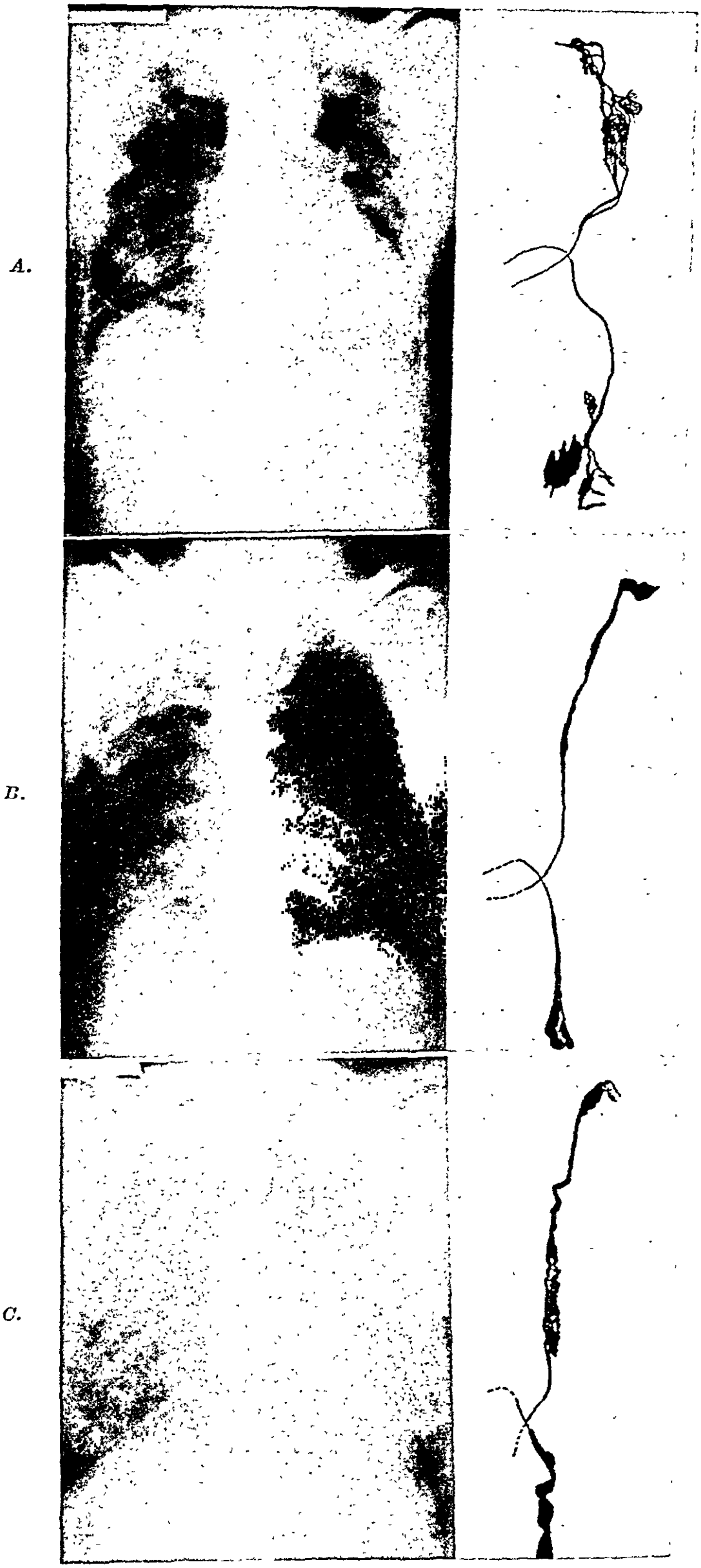


Fig. 1, *A*, *B*, and *C*. *(For legend see opposite page.)*

introduced by means of small plastic catheters with which both ends of the divided duct had been cannulated, and after the injection of 5 to 10 c c of dye both cephalad and caudad, one or more radiographic exposures in anteroposterior projection were obtained In 27 cases these studies were performed on fresh post mortem subjects by utilizing 50 per cent sodium iodide solution In the remaining 3 cases, observations were made at the time of surgery by an essentially similar technique save that 70 per cent Diodrast or Urokon was utilized Following anesthetization, the patients were placed in either the

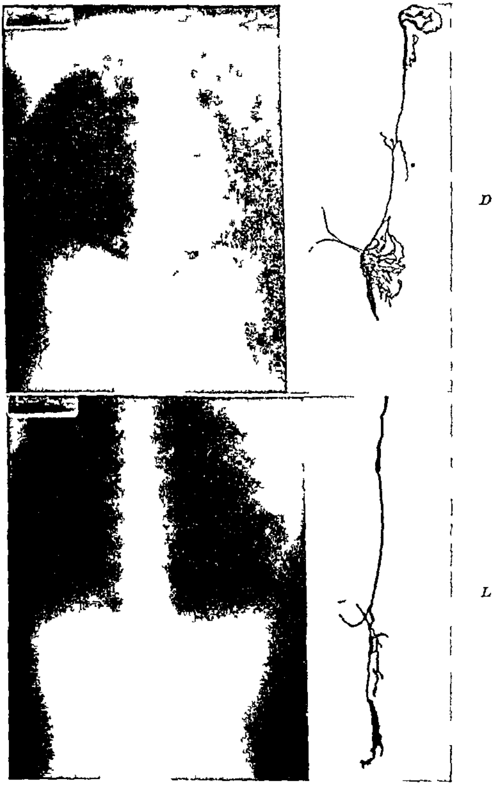


Fig 1 — Anteroposterior films of autopsy specimens following proximal and distal injection of 50 per cent sodium iodide solution into the thoracic duct at the level of the diaphragm Line drawing of duct system is to right of corresponding film *A* Complete right sided drainage. Insula in midthorax Single trunk at diaphragm Group I *B* Single duct throughout thorax Group II *C* Left sided drainage Insula in midthorax Single trunk at diaphragm Group III *D* Many trunks at diaphragm Low insula Group IV *E*, Double duct at diaphragm Group V

supine or lateral position over a portable Potter-Bucky diaphragm and exposures made immediately following injection of the dye. In 2 operative cases (Cases 1 and 2), methylene blue was injected prior to the radiopaque dye in an effort to identify the site of chyle leakage. In all the autopsy specimens, methylene blue was injected after the x-ray exposures were taken in order to facilitate identification and dissection of the duct system. The ducts were then dissected out following removal of the thoracic and abdominal viscera. Histologic examination of the cisterna chyli and various portions of the duct were undertaken and correlated with the over-all pathologic findings.

ANATOMIC FINDINGS

A brief review of the literature concerning the anatomy of the thoracic duct seems indicated at this time since diametrically opposed observations led us to pursue additional anatomic studies. That the thoracic duct is subject to considerable variation has long been recognized. Indeed, Winslow[22] in 1775 noted that the thoracic duct "is sometimes double, one on each side, and sometimes accompanied by pampiniform appendages." Rouviere[14] also noted the many variations reported in the literature and stated that articles dealing with its variations exceed those devoted to its "normal" anatomy. Davis,[6] in 1915, carefully studied the thoracic duct in 22 cases and described many variations which in conjunction with those previously reported by others led him to classify such variants into general categories which he felt were not only explainable but predictable on the basis of the embryology of the thoracic duct system which is early represented as a bilaterally symmetrical system and thus potentially bilateral throughout its entire course. Of special interest is his finding of two or more major ducts at the level of the diaphragm in approximately 33 per cent of cases. In contradistinction to these observations are those of Van Pernis[21] who, in a study of 1,081 autopsy specimens, categorically stated that the thoracic duct was always represented as a single duct below the level of the eighth thoracic vertebra. The results of Celis and Porter[5] utilizing a technique similar to the one employed in the present study, tend to support the findings of Van Pernis, although it is to be noted that in only two instances was the duct injected in a retrograde manner at the diaphragm, all others being injected only in a cephalad direction. In our opinion, this factor vitiates their findings since the roentgenographic recognition of double or collateral channels at or below the diaphragmatic level requires such retrograde filling to demonstrate their presence.

In the 30 cases studied by us, the anatomic variations could be classified according to five general groups which correspond closely to the categories of Davis. In only one instance was the duct entirely right-sided (Group I). In 9 cases the duct was essentially single throughout its course (Group II). In 10, the duct was single at and below the level of the tenth thoracic vertebra, although there was a double duct or "insular" pattern in the upper thoracic region (Group III). In 5, many channels were noted at the diaphragm with an essentially single representation above. These we have classified as having a low insula (Group IV). In the remaining 5 there were two distinct ducts at and below the level of the tenth thoracic vertebra (Group V). Representative examples of these five categories are illustrated in Fig. 1 and a summary of the anatomic findings is depicted diagrammatically in Fig. 2.

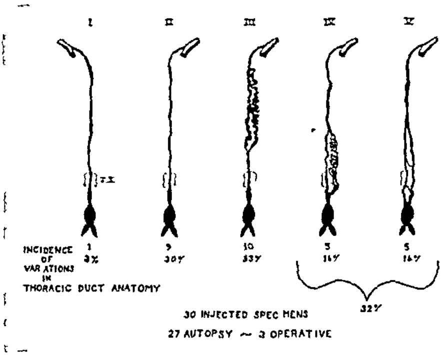


Fig. 2.—Summary of anatomic findings in thirty injected specimens classified into five general groups.

In order to assess a possible causal relationship between the variations noted and malignant disease, the pathologic diagnoses, available in all cases, were correlated with the anatomic findings. It was found that in the first three anatomic groups totaling 20 subjects, 10 (50 per cent) had neoplastic and 10 nonneoplastic disease. Of the former, most cases were far advanced with massive generalized metastases causing death. In Groups IV and V consisting of 10 subjects, 2 (20 per cent) had neoplastic disease, and the remaining 8 (80 per cent) died of nonneoplastic disorders. Continuing study of the anatomy and pathology of the thoracic duct is still in progress and will form the basis for a more detailed report in the future.

CONCLUSIONS

From the foregoing observations, certain pertinent conclusions seem inevitable. First, that contrary to the findings of some[21] the thoracic duct may be represented at the diaphragmatic level by two or more major trunks. Second, that evidence thus far does not suggest that malignancy, even though widely disseminated, is necessarily the cause of such variations, since 80 per cent of the double duct systems at low levels were not associated with malignant disease. That malignancy may cause some increase in lymphatic channels is suggested in 1 case of lymphoma (Case 3) discussed below.

Our observation that roughly one third of thoracic ducts have two or more trunks at the level of the diaphragm is obviously of practical significance in the surgical treatment of chylothorax, and a satisfactory method of recognizing such ducts at the time of operation would appear to be of distinct value. The following case reports are presented as examples of a satisfactory method to achieve this end.

CASE REPORTS

CASE 1.—G. A. S., a 57-year-old white woman, was first admitted to the Albany Hos-
tal on Nov. 12, 1952, with a complaint of increasing dyspnea of seven months' duration,
sociated with low-grade fever, but no weight loss. For three months prior to admission,
e patient had had weekly thoracenteses with relief of the dyspnea but no apparent abate-
ent in the rate of fluid formation. Physical findings revealed a thin, emaciated, chronically
l-appearing white woman, whose vital signs were within normal limits. She was moderately
yspneic and orthopneic. Significant findings were limited to the thorax where there was
idence of bilateral pleural fluid, and to the neck and axillae where palpable nodes were

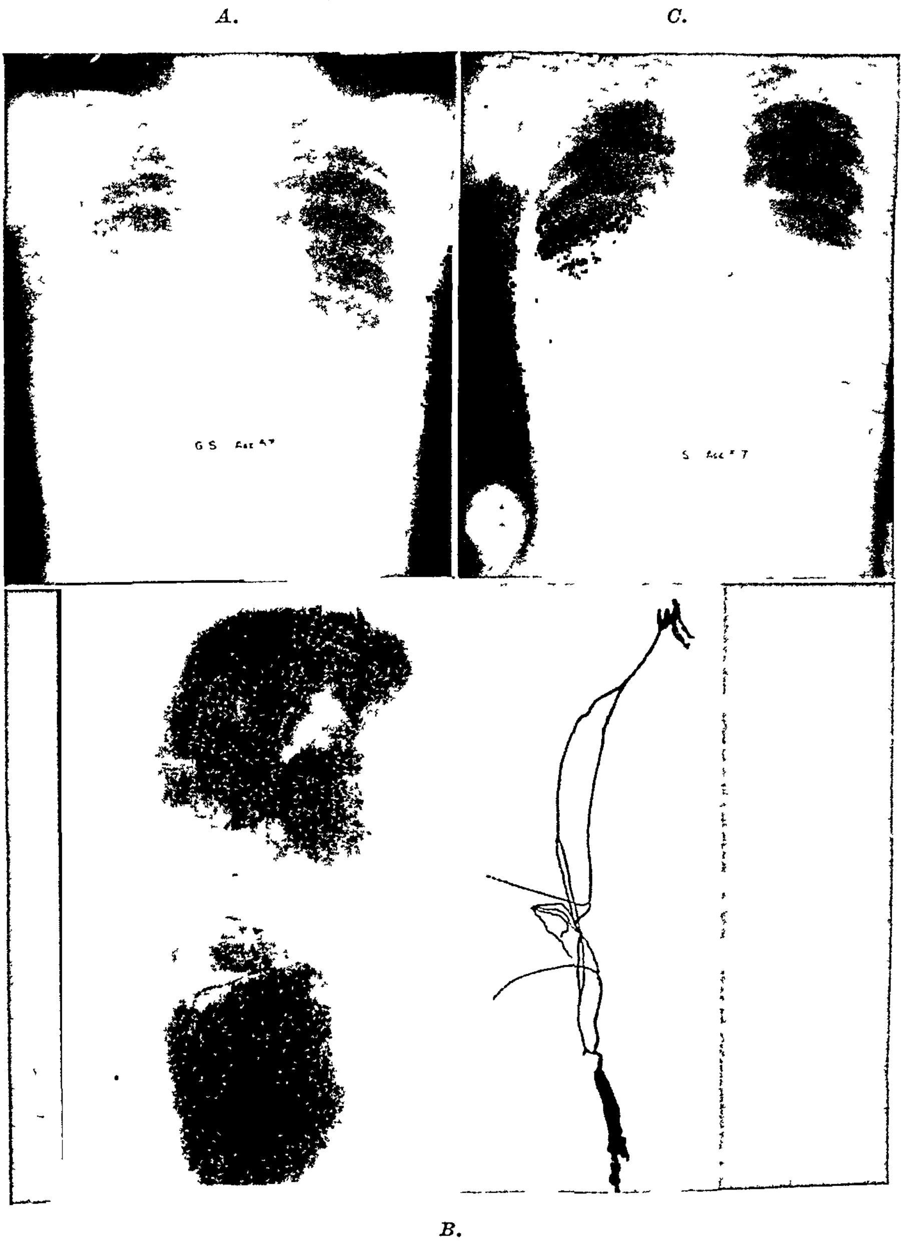


Fig. 3.—Case 1. Bilateral chylothorax in 57-year-old woman with lymphoma. *A*, Pre-
operative chest film. *B*, Operative thoracic ductogram demonstrating double duct throughout
horax. *C*, Film at discharge.

present Urinalysis was not remarkable Hemoglobin was 15 5 Gm , red count, 4 7 million, hematocrit, 45, white count, 14,300, with a normal differential Total protein was 4 5 Gm , 2 4 Gm of albumin, 2 1 Gm of globulin A cervical node biopsy revealed malignant lymphoma of the lymphocytic type Thoracenteses yielded 1,000 c c of typical chylous fluid from the right chest and a similar amount from the left The patient was given a four day course of intravenous nitrogen mustard therapy and discharged with no demonstrable benefit She was readmitted on Dec 22, 1952, because of increasing dyspnea Physical findings were essentially unchanged on this admission A chest film revealed increasing effusion bilaterally (Fig 3, *A*) The total protein had declined somewhat, being 3 6 Gm with albumin of 1 9 Gm and a globulin of 1 7 Gm As a palliative procedure, thoracic duct ligation was recommended and the patient had an exploratory operation performed on Dec 27, 1952, through the bed of the right ninth rib Upon opening the pleural cavity approximately 200 c c of chylous fluid was removed The thoracic duct was identified above the diaphragm, transected, and both ends cannulated with polyethylene catheters Methylene blue was injected proximally and distally in order to demonstrate the presence of a leak, but none was noted Following this, 70 per cent Diodrast was similarly injected and a portable film obtained The film demonstrated an accessory duct arising at the cisterna chyli with several cross connections with the cannulated duct (Fig 3, *B*) This accessory duct was located, doubly ligated above and below, and the remaining duct was then ligated proximally and distally after the catheters had been removed Following this, a decortication of the middle and lower lobes was carried out because of rather marked thickening of the visceral pleura Postoperatively the patient had no farther effusion into the right chest, although persistent chylous effusion requiring thoracenteses occurred on the left side The patient was treated by x ray to the left pleura and mediastinum and intrapleural nitrogen mustard (Fig 3, *C*) She was discharged April 19, 1953, and continued well until September when she gradually began losing ground and died on Nov 22, 1953 Permission for post mortem examination was not obtained Following discharge until the time of death the patient remained free of dyspnea and required no additional thoracenteses

Comment—The surgical result in this instance was only partially successful Chylous effusion, though controlled on the right, persisted, though to a lesser degree, on the left This is construed as confirmatory evidence that chylothorax secondary to malignancy does not necessarily indicate rupture of the thoracic duct or a major radicle thereof, but probably results from extensive disseminated pleural "weeping" of chyle The radiopaque injection of the duct at surgery resulted in excellent visualization and indicated another channel which was then located and ligated Without this aid it is doubtful that this would have been accomplished

CASE 2—J A, an 18 year old white girl, was first admitted to the Albany Hospital on June 16, 1954, because of a painless swelling, of six months' duration, in the lower left aspect of the neck On admission blood pressure was 130/70, pulse 100, respirations 20, temperature 98 6° F Significant physical findings were limited to the neck where there was a nontender nodule palpable in the left lobe of the thyroid A chest film was negative Urinalysis and a complete blood count were within normal limits On the day following admission the thyroid gland was explored at which time a cherry sized nodule was noted *in the left lobe of the thyroid and several suspicious cervical lymph nodes observed* A frozen section of one of the cervical nodes was reported as metastatic papillary adenocarcinoma in lymph node and accordingly a total thyroidectomy was performed The patient's immediate postoperative course was uncomplicated and two days later, under general endotracheal anesthesia, a re exploration was performed, with a left radical neck dissection and sternal split with lymph node dissection of the superior mediastinum[12] During the procedure the left pleural cavity was entered, and the cervical thoracic duct was ligated and removed with the specimen Catheters were placed in the left pleural space and superior mediastinum for drainage The patient's postoperative course was uncomplicated and both catheters were removed at the end of forty-eight hours. Fluoroscopy shortly prior to discharge on June 19 revealed excellent expansion of the left lung with no effusion in either pleural cavity.

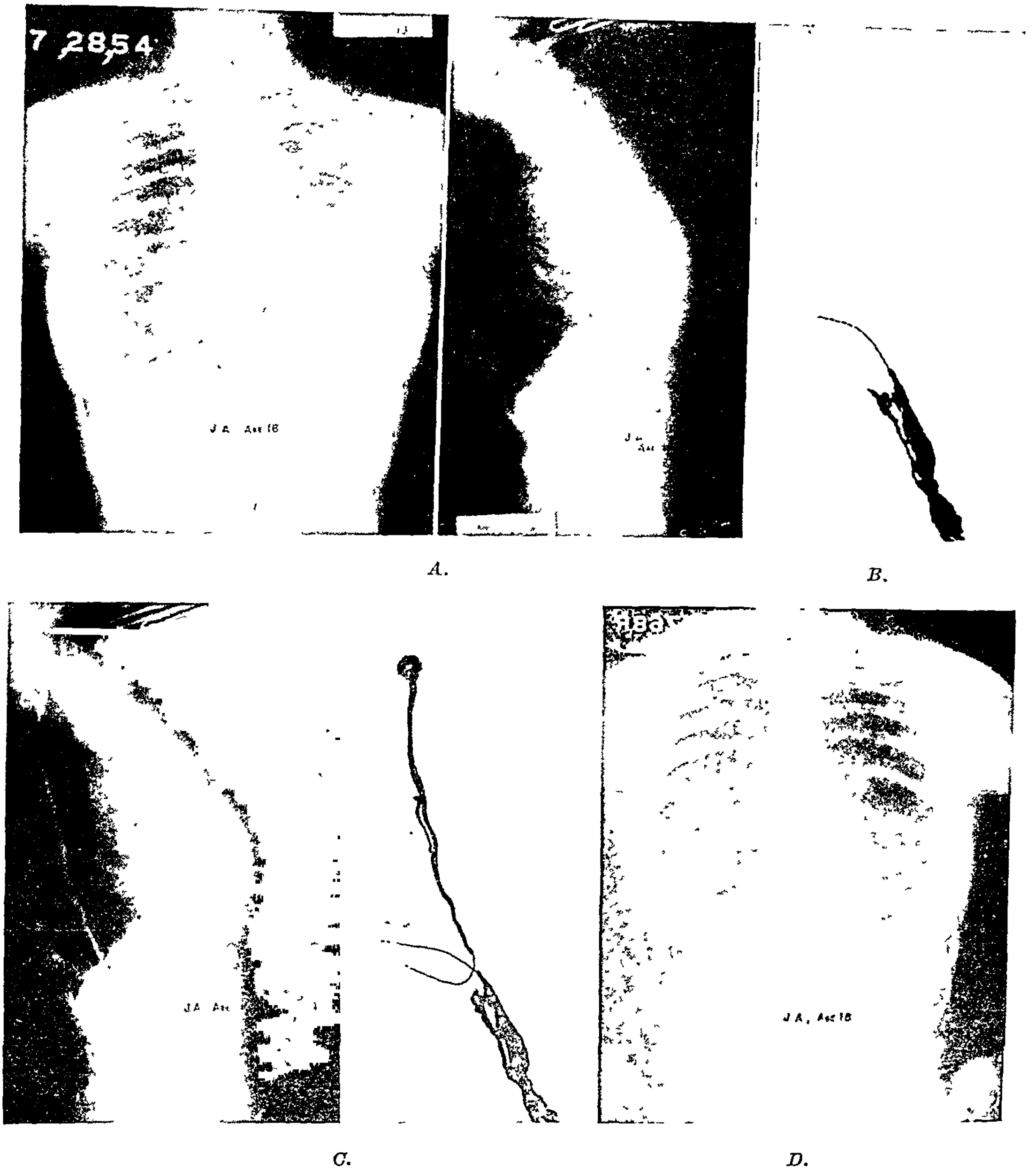


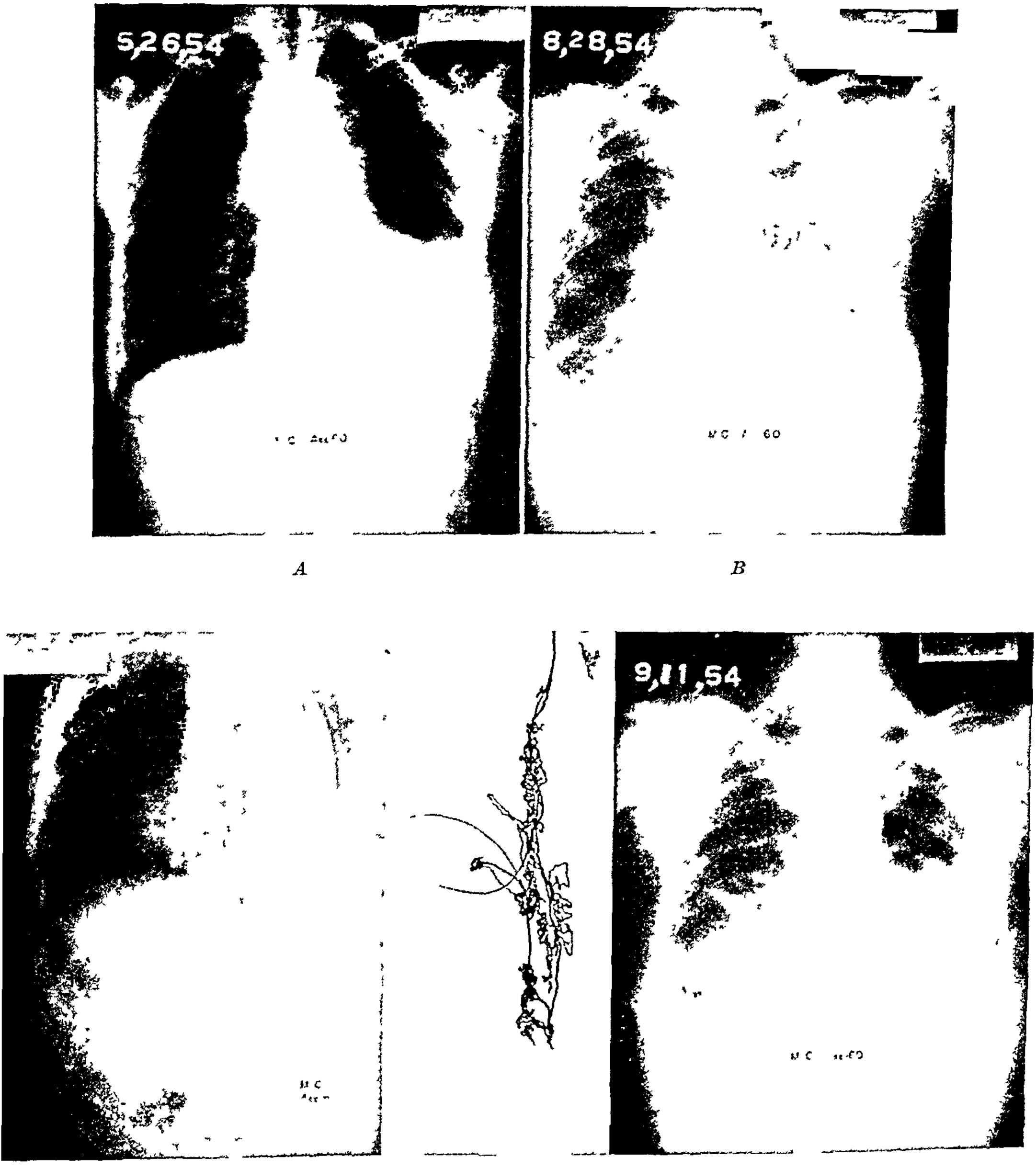
Fig. 4.—Case 2. Traumatic chylothorax in 18-year-old white girl following sternotomy and neck dissection for thyroid cancer. *A*, Left chylothorax on second admission seven weeks after initial operation. *B*, Operative injection of thoracic duct distally. *C*, After proximal and distal injection. There is a single duct at the diaphragm, a short double duct in midthorax. *D*, Chest film six months after ligation of thoracic duct at diaphragm.

When she was seen two weeks following discharge there was noted, fluoroscopically, a small pleural effusion at the left base which was not considered significant. Three weeks later the patient complained of marked dyspnea and on fluoroscopy there was shift of the mediastinum and heart to the right with a massive left-sided pleural effusion (Fig. 4, *A*). Thoracentesis yielded 1,500 c.c. of chylous fluid which re-accumulated rapidly in a matter of four days and accordingly the patient was readmitted to the hospital on July 27, 1954.

Physical examination revealed the vital signs to be within normal limits The [illegible] and sternotomy incisions were well healed and the left hemithorax was flat to percussion with diminished to absent breath sounds throughout An intercostal catheter was inserted into the left pleural cavity and 1,200 c c of chyle obtained The patient was then maintained on catheter drainage but during the ensuing thirteen days there was no abatement in the amount of drainage Serum electrolytes at this time were within normal limits Total protein was 6 3 Gm with 3 9 Gm of albumin and 2 4 Gm of globulin On Aug 9, 1954, an exploratory operation was performed through the seventh left interspace at which time 1 600 c c of chyle were aspirated from the left hemithorax There was no thickening of the visceral pleura, although there were adhesions between the upper lobe and superior mediastinal pleura The aorta was mobilized just above the diaphragm by ligation and division of two pairs of intercostal arteries and retracted anteriorly with a Penrose drain *A somewhat enlarged thoracic duct was easily identified, and following division at this level* both ends were cannulated with small plastic catheters Several cubic centimeters of *methyl*ene blue were injected both proximally and distally into the thoracic duct *through the* catheters and extravasation occurred into the mediastinum at the apex of the thorax and somewhat to the left of the subclavian artery No definite isolated area of leakage was noted but it appeared to be a diffuse process Five cubic centimeters of 70 per cent *Diodrast* was injected into the distal catheter and an x ray taken (Fig 4, *B*), following which a *similar* amount of Diodrast was injected proximally and another x ray obtained (Fig 4, *C*) *There* was no evidence of a second thoracic duct and accordingly the catheters were withdrawn and the proximal and distal ends of the thoracic duct triply ligated with No 2 0 *silk* The pleural cavity was copiously irrigated with saline and the underlying lung re expanded The pleural space was drained with two separate catheters and routine closure of the wound carried out Following this procedure the patient's postoperative course was uncomplicated and she was discharged on Aug 17, 1954, on the eighth postoperative day The patient *has* subsequently done well and at no time has there been any evidence of re accumulation of chyle in the chest A film obtained approximately six months following ligation of the thoracic duct reveals an essentially normal chest (Fig 4 *D*) There is no evidence of *further thyroid malignancy*

Comment—Although the cervical thoracic duct was identified and ligated at the time of radical surgery on the neck, an unrecognized trunk was probably injured That the chylothorax resulted from rupture secondary to ligation is highly unlikely in view of experimental evidence[4 10] and in view of the findings at the time of low ligation of the duct Demonstration of a single duct by contrast roentgenology at the time of ligation virtually assured a successful result Approach to the duct from the left side in this case was greatly facilitated by anterior retraction of the aorta after division of two pairs of intercostal vessels

CASE 3—M C, a 60 year old white single woman, was admitted to the Albany Hospital May 18, 1954, with a chief complaint of abdominal pressure, backache, malaise, and fatigue, of approximately three months duration, associated with a 10 pound loss of weight For two weeks prior to admission there had been increasing exertional dyspnea Physical findings revealed a pale, chronically ill woman with obvious weight loss Temperature 99° F, pulse 120 respirations 24, blood pressure 120/70 Supraclavicular and axillary adenopathy were noted There was dullness with diminished breath sounds over the left chest posteriorly The liver was palpable two fingers below the right costal margin and there was a large nontender mass in the epigastrium Urinalysis was negative A blood count revealed 11 2 Gm of hemoglobin, with a red count, 3 6 million, hematocrit, 39, white count 5 400 with a mass and a left sided pleural effusion (Fig 5, *A*) A diagnosis of malignant lymphoma normal differential Films of the chest revealed the presence of a superior mediastinal lymphoblastic type, was made by biopsy of a supraclavicular lymph node and the patient was treated with a four day course of intravenous nitrogen mustard followed by x-ray therapy to the mediastinum, abdomen, and axillary areas for a total of 2,000 r tumor dose during May, June, and early July. Objectively there was fairly good response to this regimen with disappearance of the regional adenopathy, the epigastric mass, and the mediastinal tumor



A B

C D.

Fig. 5.—Case 3. Chylothorax from lymphoma in 60-year-old woman. *A*, Chest film on admission showing mediastinal mass and left chylous effusion. *B*, Chest film three months later following x-ray therapy and two courses of nitrogen mustard. Mediastinal nodes have regressed but effusion has persisted and increased. *C*, Operative ductogram after injection. A second major duct is apparent by retrograde filling from the cisterna chyli. Both ducts shown connecting with midthoracic insula. *D*, Chest film nine days following interruption of duct. There is marked clearing but still persistent fluid with some air. Pleural reaction probably prevented full expansion of left lung.

on follow up x ray examination, however the patient's complaint of dyspnea increased and it was necessary to perform thoracenteses with increasing frequency so that in the ensuing three months approximately 15 L of chylous fluid had been aspirated from the left hemithorax In August of 1954 the patient was again given a four day course of nitrogen mustard therapy without appreciable benefit and because of a rather marked depression of the white count, additional x ray therapy was not deemed advisable Since, at this time, the patient's symptoms were exclusively related to her effusion requiring thoracentesis once and frequently twice a week for relief, ligation of the thoracic duct as a palliative procedure was considered (Fig 5, *B*) Following several transfusions to improve the patient's anemia she was taken to the operating room on Sept 2, 1954, where, under general endotracheal anesthesia, left sided intercostal catheter drainage was established with the immediate withdrawal of 1,500 c c of chylous fluid the catheter then being attached to an under water seal The patient was then placed over a portable Bucky x ray diaphragm, positioned in a supine position with slight elevation of the right chest and the table tilted 15 degrees to the left to elevate farther the right chest A submammary incision was made from the axilla medially to expose the eighth interspace which was incised with transection of the costal arch No fluid was encountered in the right hemithorax The right lung was free of adhesions to the diaphragm or parietal pleura Pulmonary ligament was partially transected and the right lung mobilized and retracted upward and out of the way The diaphragm was retracted inferiorly and the mediastinal pleura entered just medial to the azygos vein Tissues in this area were indurated and appeared to contain tumor in masses of nodes It was feasible to identify a single large duct which was transected, catheterized distally and proximally with plastic catheters, following which 5 c c of 70 per cent Urokon was injected into the distally placed catheter and a film obtained, following which 10 c c was injected into the proximal catheter and an additional film taken (Fig 5, *C*) The x ray films indicated that the entire area above the diaphragm appeared to be composed of one accessory and numerous collateral ducts rather than the single duct which had been cannulated Accordingly, after simple ligation of the major duct, transfixion sutures were placed just medial to the aorta through the anterior longitudinal ligament and tied The area was packed with Oxycel gauze to promote fibrosis, following which the mediastinal pleura was sutured the right lung re expanded and a catheter left in the right chest The patient's postoperative course was essentially uncomplicated and both catheters were removed by the third postoperative day On the sixth postoperative day, a thoracentesis was performed on the left side and 500 c c of chyle was obtained It was felt that this was residual and did not necessarily represent re formation At the time of discharge on the patient's ninth postoperative day she was ambulatory and afebrile on x ray examination the right chest was free of fluid and the left chest contained some fluid and incomplete expansion of the left lung (Fig 5, *D*) Following discharge the patient did fairly well but gradually continued on a downhill course and was readmitted for terminal care dying on Oct 8 1954 Although this patient survived surgery for only a period of slightly over five weeks, during that time she was clinically free of dyspnea and required no additional thoracenteses Even on her final admission, breath sounds were clear bilaterally At autopsy, generalized lymphoma involving the cervical supraclavicular, axillary, inguinal mediastinal aortic, mesenteric and hilar nodes was noted as well as extensive involvement of the left parietal pleura and pelvic peritoneum There was minimal fluid in the right chest and 300 c c of chyle in the left chest

Comment—Thoracic ductography at operation indicated that simple ligation of the cannulated duct would probably have been ineffective It is felt that in this case the many collaterals resulted from the underlying disease Though survival was brief following surgery a satisfactory result with regard to marked decrease in chylothorax was apparent, The right submammary approach was utilized since it was considered to be a more rapid and less traumatic route in this debilitated patient. The extensive left-sided mediastinal and pleural involvement by tumor noted at autopsy would have made a left-sided approach to the duct impossible.

DISCUSSION

The surgical management of traumatic chylothorax is well established, and though all observers recommend early conservative management, recent reports[1, 16] indicate a more aggressive attitude favoring earlier surgical intervention when it becomes apparent that lesser measures are ineffective. In general, simple ligation of the duct at diaphragmatic level is definitive and the procedure of choice. Other technical methods including plastic repair of the duct as advocated by Seaman[17] or implantation of the duct into the azygos vein as proposed by Hodge and his associates[8] are based upon the repudiated premise that ligation will result in rupture of the duct, are technically more difficult and time consuming, and are certainly not applicable in all instances. We concur with those who favor exploration on the side of the chylothorax in traumatic cases, since complete evacuation of chyle from that hemithorax is possible and decortication can be accomplished if indicated. Traumatic chylothorax on the left subsequent to esophageal resection with left-sided esophagogastrostomy is probably an exception.

Though less frequently encountered, spontaneous chylothorax not the result of neoplasm is also amenable to surgical correction and presents essentially the same problems in management as do the traumatic cases. In chylous effusion secondary to malignancy, which is relatively frequent, little information can be gained from the literature. Meade and his associates[13] reported a case in which exploration was performed twice for spontaneous chylothorax and, though malignancy was suspected, it was not proved until later. Deliberate surgical intervention for chylothorax secondary to neoplasm is at best palliative, and should be undertaken only when all other modalities of therapy, which are frequently effective in controlling such chylous effusion, have been attempted. Our results in the surgical treatment of two such cases are not striking but do suggest further evaluation and trial in selected cases.

Operative visualization of the thoracic duct system by the technique described is relatively simple, adds little to the operative time, and is felt to be of value in assessing the presence or absence of other trunks which may then be located and ligated. Since such variations are present at diaphragmatic level in approximately 30 per cent of the instances, the information gained is of obvious importance. Where lesions such as cysts or hygromas of the thoracic duct are undiagnosed even at exploration the method can be of distinct diagnostic value, and its routine employment is recommended.

SUMMARY

1. Anatomic studies of the thoracic duct system in 30 subjects by means of contrast roentgenography are presented.

2 A simple method of visualizing the entire thoracic duct system at surgery is described and 3 cases of chylothorax in which such operative thoracic ductograms were obtained are reported

3 The surgical management of chylothorax is discussed and intervention in selected cases due to malignancy as a palliative procedure is suggested

4 Routine use of operative thoracic ductography is recommended in operations for chylothorax

The authors wish to express their thanks and appreciation to Dr Arthur W Wright Professor of Pathology, Albany Medical College, for permission to carry on the anatomic studies in his department, and also to Dr John F Roach, Professor of Radiology, Albany Medical College, for his technical assistance and cooperation in this project

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DISCUSSION

DR ELMER P R MAURER, Cincinnati, Ohio —The amazing adequacy of the collateral circulation about the thoracic duct was impressed upon me in May, 1953, when I was asked to see a 37year old white woman with fluid in the right chest

(Slide) I think you can see the accumulation here Thoracentesis yielded 1,000 c c of creamy odorless fluid which had the characteristics of chyle This was alkalinized and treated with ether This was before this method of treatment and after, giving the characteristic reduction in volume The patient was treated with multiple thoracenteses and closed tube thoracostomy drainage for a period of three weeks with removal of approximately 7 5 L of chylous fluid without any improvement

(Slide) On June 15, 1953, a right thoracotomy was performed through the eighth rib bed. You can see a cylinder-shaped tumor mass involving the thoracic duct, measuring approximately 2 cm. in diameter, situated between the azygos vein and the aorta. Chyle leaked constantly through pin-point openings in this tumor. As the dissection was continued it was found that the tumor involved the entire thoracic duct from the aortic hiatus up to the point where it crossed the midline into the opposite side and the thoracic inlet. The duct in the normal portion high underneath the left pleura was ligated and again inferiorly above the aortic hiatus, following which the entire tumor and thoracic duct were removed.

(Slide) Microscopically, the tumor resembled the characteristic picture of benign lymphangioma with numerous networks of irregular endothelium-lined channels containing lymph.

(Slide) The patient did well postoperatively and was ready to go home on the eleventh day. There was no recurrence of the chyle and, up to the present time, approximately two years after operation, she has done well.

To my knowledge this is the first case of primary benign tumor of the thoracic duct that has been reported in association with spontaneous chylothorax. Also, in a review of the literature I have encountered no instance of excision of extensive portions of the thoracic duct for any cause whatsoever. Obviously, as demonstrated as early as 1922 by Ferdinand C. Lee, the thoracic duct can be ligated with impunity and, following ligation (and I think this is important) collateral lymph circulation is established through embryonic lymph channels which normally do not function, which in response to ligation may be forced to enlarge and dilate. Also, following ligation lymphatico-venous connections are found to exist between the thoracic duct, the azygos, intercostal, and lumbar veins. The embryonic thoracic ducts are bilateral, symmetrical, and have numerous cross anastomoses. Despite the excellent demonstration Dr. Stranahan and his associates have presented today, I think the factor of excellent collateral circulation, particularly in light of the clinical experience I have just reported, would indicate that extensive resection of the thoracic duct is possible even in those patients who have a single canal, as reputedly occurs in some 60 per cent of the cases.

DR. ROY KLEPSER, Washington, D. C.—It is surprising how many surgeons operating in the neck, mediastinum, and upper abdomen, within a few centimeters of the thoracic duct, have never taken the trouble to expose it and to look at it, much less to wonder what they would do if they did cut it.

In 1949, while discussing the paper of Dr. Meade at the New Orleans meeting, I presented a method of diagnosis of chylothorax with the aid of lipophilic dyes. My purpose in devising the test was to outline the thoracic duct for verification at the time of operation. This did not work well as the dye was not concentrated enough. It was a rather pastel color in the duct, and the duct did not visualize because of the pale color in the rather opaque duct. However, the fat in which the dye was fed to the patient distended the duct, making it easily identifiable. If one gives the patient a small amount of olive oil or butter preoperatively, the duct can be seen to be the size of a lead pencil instead of the size of the lead of a pencil. We are used to seeing the duct in a collapsed state in the cadaver, or in the starved state in which we operate on patients, so we seldom see a distended duct. The duct thus becomes much more readily identified if it is distended with oil preoperatively.

In using the dyes diagnostically the dye is mixed with butter and is fed to the patient the night before thoracentesis. It is better to feed it as a sandwich, in the dark, because the dyed butter looks like used wagon grease. It is spread thickly, about a quarter pound of butter with an ounce of the dye. The following day the chest is tapped. The chyle, which has been milky or possibly blood-tinged, will then be green, or purple, or red, depending on the dye used. This test not only tells us that it is chyle, but also tells us that the duct is still leaking.

Our method of ligation is to go in on the right side through an anterolateral incision between the ribs, ligating the duct above the diaphragm. I have ligated twenty-seven thoracic ducts in the past five years. Nine ligations were for ruptured thoracic ducts. The other eighteen ligations were in unresectable carcinoma cases, the ducts being ligated while the chest was open in order to evaluate the effect of its ligation. In no cases were there harmful effects from this.

DR. LYMAN A. BREWER, III, Los Angeles, Calif.—I think the essayists have presented a real contribution because the location of the duct and its tributaries is sometimes most important. We have recently reviewed a series of 24 cases of thoracic duct lesions of various types. This report will be published in the *American Journal of Surgery* in the near future. In the cases studied in our series, the most common source of leakage of chyle from disease is not in the thorax, but in the abdomen. Therefore the failure of treatment of chylothorax by ligation of the duct or other procedure may be explained by the fact that the chyle is extravasated below the diaphragm and leaks up into the chest along the paravertebral space to seep out into the mediastinal pleura to the thorax. The chyle may also extravasate down the leg or up into the cervical region or even out into the arms. Therefore no exploration is complete without a search for the pathology in the abdomen or retroperitoneal tissues. We believe that it is important that the surgeon recognize the wide collateral circulation of the thoracic duct system, for this permits ligation of the thoracic duct in the neck and in the thorax and, in most instances, in the upper abdomen above the cisterna chyli. The lesions below this level, which are primarily obstructive in nature, present a more complex problem.

We have recently reported a case of a neurogenic tumor involving the thoracic duct at the cisterna chyli. In this case the subdiaphragmatic collection of chyle worked its way along the paravertebral space and through the various diaphragmatic hiati and into the pleural cavity. This was discovered for the first time when the thorax was explored to determine the cause of a persistent spontaneous chylothorax. When the chyle was obviously coming up from below the diaphragm, this structure was opened to reveal the true source of the disease. After excising the neurogenic tumor which had caused the obstruction of the afferent ducts to the cisterna chyli, we found that there was a dilatation of one of the major ducts which, after ligation, was on the point of rupturing. It was obvious that decompression of this duct was necessary, for we had merely reproduced the obstructive phenomena of the tumor. Therefore a side-to-side anastomosis of the thoracic duct to the hemi-azygos vein was successfully completed. There has been no recurrence of the chylothorax or drainage of chyle below the diaphragm for over three years, pointing to the fact that the anastomosis must still be patent. We believe the principle of decompression of the thoracic duct, which has ruptured as the result of obstruction, is sound. It probably has application only in the abdominal portion of the duct and, from the clinical and experimental evidence at hand, most often in the afferent branches to the cisterna chyli. Since the abdominal lesions were most common in the series that we have studied, further application of this technique will be indicated. The manner of identification of the branches of the thoracic duct presented by the authors is of considerable importance, for it may make possible the performance of thoracic duct venous anastomoses in certain cases.

DR. W. O. MILLS, Seattle, Wash.—I have enjoyed this presentation very much, and the beautiful contrast medium studies presented are certainly helpful in visualizing the often plexiform nature of the thoracic duct channels. However, I think we ought to remember that our success in ligating such a complex structure may not be as high as we think at the time we see the operation. We should also remember that despite the quality of our visualization and the delicacy of our dissection, one of the things that controls chylothorax is obliterative pleuritis. In each of the cases presented today, whether the chylothorax is bilateral or unilateral, the benefit from the operation lies on the side that was operated upon. In each instance the costophrenic angle was flattened and there is evidence of obliterative pleuritis on the operated side. It may be that this has contributed to the good control of the chylothorax. As far back as 1943, I remember a right chylothorax due to a gunshot wound that was treated by aspiration of the chyle and intrapleural injections of glucose solution. We got a very prompt and good result. I believe that often the obliterative pleuritis is the thing that controls the situation.

DR. KAUSEL (Closing).—My associates and I would like to thank the discussants for their remarks. With regard to Dr. Maurer's comments, I think other cases of hygromas or lymphangiomas of the thoracic duct have been reported; Dr. Brewer's case certainly sounds like one and I think Drs. Touroff and Seley reported such an instance. We are well aware of Dr. Klepser's work with lipophilic dyes and we concur that they are of great diagnostic value. We did not discuss diagnosis in this paper.

Dr. Brewer's remarks about associated chylous ascites are quite true. We have seen such a case of chylous ascites associated with chylothorax. We did not operate on this patient, the ascites and effusion being controlled by other means. With regard to Dr. Mills's discussion, I believe his observations about the efficacy of the obliterative pleuritis are quite true; for these reasons I think that in traumatic cases the approach should be through the side of the chylous effusion. At this time the chest can be completely exacuated of chyle and if necessary, and not infrequently it is, the lung can be decorticated. This then lends itself to a good pleuritis. However, I have one reservation; that is, in those cases with apparently adequate tube drainage, where the leak persists for two or three weeks and, following ligation of the duct, stops, obliterative pleuritis is not the entire answer—I do not know the entire answer to it, and I think it is imporant.

We would like to make one other comment, with regard to intervention in cases of malignancy. We do not recommend this as a routine measure and the majority of the cases we have seen have responded to intravenous nitrogen mustard and x-ray, or intrapleural nitrogen mustard, and obviously those modalities should be tried first in cases secondary to malignancy.

DR. EDWARD S. WELLES, Saranac Lake, N. Y.—I have been very quiet throughout this entire meeting, but at this time I want to compliment those who discussed this paper. All the discussions have been pertinent to the subject and have dealt with the thoracic duct. I should like to call to the attention of the younger men particularly the fact that this is the type of discussion we ought to have. So many young men look over a program and find a subject that has some vague connection with what they want to say, so they take the opportunity to appear on the platform with a lot of statistics and present what is really a paper, although it would not be accepted as such, which has merely some faint relationship to the subject at hand. Therefore, I want to go on record as complimenting all the men who discussed this paper.

Editor's Note

It is to be regretted that the next paper by Dr. Charles B. Ripstein of New York, entitled "Complications of Mitral Valve Surgery With Particular Reference to the Post Commissurotomy Syndrome," was not turned in for publication. Efforts to obtain the paper were unsuccessful. Under the circumstances it seemed of no value to publish the interesting discussions of it which were given by Dr. Charles P. Bailey, Dr. Harris B. Shumacker, Dr. Egbert H. Fell, and Dr. E. D. Cagnon.

EVARTS A. GRAHAM.

VASCULAR CHANGES IN BRONCHOPULMONARY SEQUESTRATION

OBSERVATIONS IN TWO CASES WITH MULTIPLE ACCESSORY SYSTEMIC ARTERIES

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THE occurrence of aberrant systemic arteries to the lung has been noted with increasing frequency and interest since the advent of modern thoracic surgery. While earlier reports[1-4] dealt primarily with cases encountered in the anatomy and pathology laboratories, the more recent publications have stressed the frequency and importance of this syndrome in surgical diagnosis and treatment. Several excellent recent reviews of this subject[5-11] obviate the necessity of our reiterating the existing literature at this time. Instead, we propose to summarize briefly certain aspects of the current knowledge in this field and to add to it certain observations based on detailed personal study of two cases of multiple accessory systemic arteries to the lung.

Most aberrant vessels to the lungs originate from the descending aorta, usually just above the diaphragm or sometimes just below it. While the occurrence of aberrant vessels with origins from other large systemic arteries has been reported, such arteries are more likely to be associated with anomalies of the cardiovascular system. In all the cases of accessory systemic arteries arising from the lower thoracic aorta which have been reported to date, only one aberrant artery was found to course to any one lung. Where two such vessels have heretofore been found, they entered opposite lungs. Aberrant systemic arteries may supply a lung which is otherwise entirely normal, but the majority of such vessels have been distributed to areas of the lung, most commonly the lower lobes, which also showed other abnormalities. In most cases the affected lobe or some of its segments have been the seat of multiple cysts or of marked cystic bronchiectasis. Frequently, the dilated bronchi cannot be demonstrated to have significant communications with the remainder of the bronchial tree (intralobar sequestration). Accessory systemic vessels have at times been found in association with anomalies of the diaphragm. There is general agreement that accessory systemic arteries to the lung as well as the associated pulmonary abnormalities are congenital in origin. The exact mode of origin of these changes is in doubt. Of the various theories which have been proposed to account for this syndrome,[5, 6, 8, 10, 12] Pryce's views have found wide acceptance in recent years.

The structure of the aberrant vessels was also studied by Pryce.[5] He found them to be elastic arteries and in comparing them to pulmonary arteries noted that the former were thicker walled and contained more elastic tissue

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While commenting on these quantitative differences between the two types of vessels, he apparently found no qualitative differences in their structure. He concluded that the aberrant vessels were indeed pulmonary in origin. Pryce also observed varying degrees of atheromatosis in aberrant systemic arteries and also called attention to Batts's[4] previous description of degenerative changes in such anomalous vessels. Bruwer and associates[8] also observed such degenerative phenomena and regarded them as possibly related to the patient's age and to sepsis as well as to the fact that the portion of the lung supplied by the aberrant artery is usually nonfunctioning. These authors also subscribed to Pryce's view that these vessels differ only quantitatively from pulmonary arteries. In none of these studies was there any attempt to relate the degenerative changes in the aberrant vessels to the presence or absence of such changes in the normal pulmonary vessels.

Clinically, accessory systemic arteries by themselves do not give rise to symptoms or signs. The associated pulmonary changes may be symptomatic when secondarily infected. This, as well as their discovery on survey chest roentgenograms, usually brings them to the surgeon's attention. The necessity for awareness of the occurrence of aberrant systemic arteries, especially in the performance of pulmonary resections in young individuals with cystic lesions, was emphasized by Douglass[7] and Harris and Lewis.[13] Death from exsanguination when such a vessel was inadvertently injured in the course of lobectomy has been reported.[13]

The coexistence of pulmonary abnormalities and pulmonary tumors, especially the so-called bronchial adenomas, has been repeatedly noted and emphasized by Womack and Graham.[14, 15] Until now, no tumors have been reported in lobes with bronchopulmonary sequestration and aberrant systemic arteries.

CASE REPORTS

Case 1.—(R. F., No. 53740.) The patient was a 14-year-old white boy who was admitted to the Jewish Hospital of St. Louis on April 10, 1953, with the history that he was in apparent good health until approximately four months previously when he had "flu" which was associated with chills, fever, cough, and expectoration. Under treatment by his local physician his symptoms improved rapidly. About one month later, he began to have a mild cough, productive of approximately one teaspoonful of pinkish sputum daily. On April 3, 1953, a routine survey chest roentgenogram taken at school showed a lesion in the left lower lobe and the patient was advised to consult his physician. A tuberculin patch test had been negative in January, 1953. During these months, the patient continued to feel well and took part in competitive high school sports. His past history was relevant only in that he had had mild asthma since early childhood. The last attack had occurred seven months prior to admission. Six years previously he had had pneumonia from which he made an uneventful recovery.

Physical examination on admission showed a well-developed, well-nourished 14-year-old boy who was not acutely ill. Examination of the chest revealed normal expansion. Percussion note was resonant. Breath sounds were within normal limits and there were no râles. The remainder of the physical examination was similarly unrevealing.

All laboratory studies, including urinalysis, blood counts, Kahn test, serum protein, blood sugar, and urea determinations were within normal limits. Sputa were negative for acid-fast bacilli. Tuberculin and histoplasmin skin tests were negative. Roentgenogram of the chest showed a cavitary lesion in the posterior basal segment of the left lower lobe in which a fluid level was seen. Bronchoscopy on April 13, 1953, showed a small amount of purulent material coming from the left lower lobe orifice but there was no tumor or other obstruction.

A tentative diagnosis of pulmonary cyst or chronic lung abscess was made and the patient was scheduled for operation and meanwhile discharged from the hospital on *sulfadiazine, 1 Gm., four times daily.* He was readmitted to the hospital on April 26, 1953, with no complaints. He had been asymptomatic since his discharge. Physical examination and laboratory studies were again unrevealing. A new chest roentgenogram showed no change in the appearance of the lesion.

On April 30, 1953, left lower lobectomy was performed under endotracheal ether anesthesia. At operation a cystic lesion was found in the posterior basal segment of the left lower lobe. In palpation of the inferior pulmonary ligament, pulsating vessels were felt. By careful dissection, six arteries varying in diameter from 2 to 5 mm. were successively identified and divided after ligation. Each of these vessels originated in the descending aorta about 3 to 4 cm. above the diaphragm and entered the left lower lobe below the inferior pulmonary vein through the pulmonary ligament. The remainder of the lobectomy was accomplished in routine fashion.

The patient's postoperative recovery was uneventful and he was discharged from the hospital on May 10, 1953. He has remained well since then.

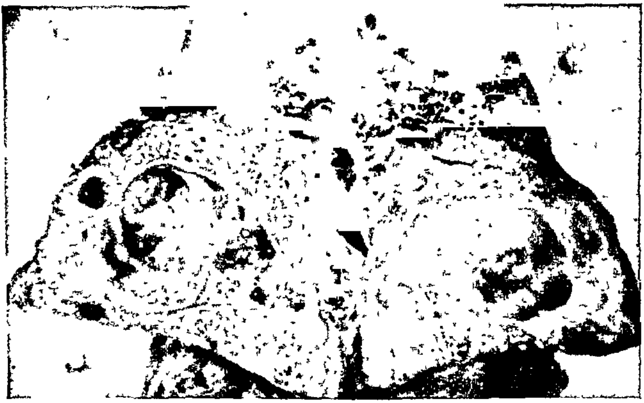

Fig. 1.—Gross specimen, Case 1. A portion of the diseased lung has been bisected showing numerous small unilocular and larger multilocular cysts

Gross Pathologic Description.—The specimen consisted of the lower lobe of the left lung. In the lobar hilum, the artery and bronchus to the basal segments as well as the separately divided apical segment artery and bronchus were identified. At the medial and posterior aspect of the lobe below the inferior pulmonary vein, six ligated arteries could be seen. They varied in size from 1 to 3 mm. in diameter and were relatively thick walled. Over the posterior basal segment, the visceral pleura showed numerous old fibrous adhesions. This area of the lobe felt firm and indurated. The abnormal part of the lobe was sharply demarcated. Under fluoroscopy Diodrast was injected into the basal segment artery of the lobe. This artery was seen to divide into two segmental branches which supplied the anterior and middle basal segments. The posterior basal segment did not receive a grossly recognizable branch and scarcely showed opacification by the dye. Injection of anomalous arteries with Diodrast was then done and resulted in visualization of a branching network of vessels. The posterior basal segment became opacified after this injection. Dissection of the bronchial tree showed two major segmental bronchi arising from the common basal segmental bronchus. These bronchi and the apical segmental bronchus skirted the indurated posterior basal segment but did not supply it except for one small branch about 2 mm. in diameter which entered this segment. On further sectioning of the lobe, the entire lobe with the exception of the posterior basal segment was found to be normal. The posterior basal segment was entirely replaced by one large cyst measuring about 5 cm. in diameter and numerous smaller cysts which varied in size from less than 1 mm. to 1.5 cm. in diameter. The previously mentioned small bronchial branch was the only recognizable communication between the large cyst and the remainder of the bronchial tree. The intervening pulmonary parenchyma was firm, grayish, and speckled with small yellowish and brownish areas (Fig. 1).

Microscopic Description.—Sections were taken from the uninvolved portion of the lobe, through the anomalous arteries and from various portions of the abnormal segment of the lobe and were stained with hematoxylin and eosin, and Verhoeff's elastic tissue stain. In addition, micro-incinerated sections were examined with the dark field technique. Sections through the posterior basal segment showed the normal architecture of the lung completely destroyed. Instead there were innumerable small cystlike spaces as well as larger cysts. The larger cysts were lined by tall columnar ciliated epithelium. The smaller cysts showed a lining which was sometimes cuboidal, ciliated, and obviously secretory, at other times low cuboidal and nonciliated. Many of the smallest cysts were partly lined by one type of epithelium and partly by another (Fig. 2). The lumen of the cysts contained mucoid material which was lightly pink staining with hematoxylin and eosin, and mononuclear and multinucleated phagocytic cells. There were also a few lymphocytes, but no appreciable number of polymorphonuclear leukocytes. Beneath the epithelium of the largest cyst there were scattered groups of smooth muscle fibers and small rounded pieces of cartilage. A few mucus glands were also present in the wall of this cyst. The intervening tissue

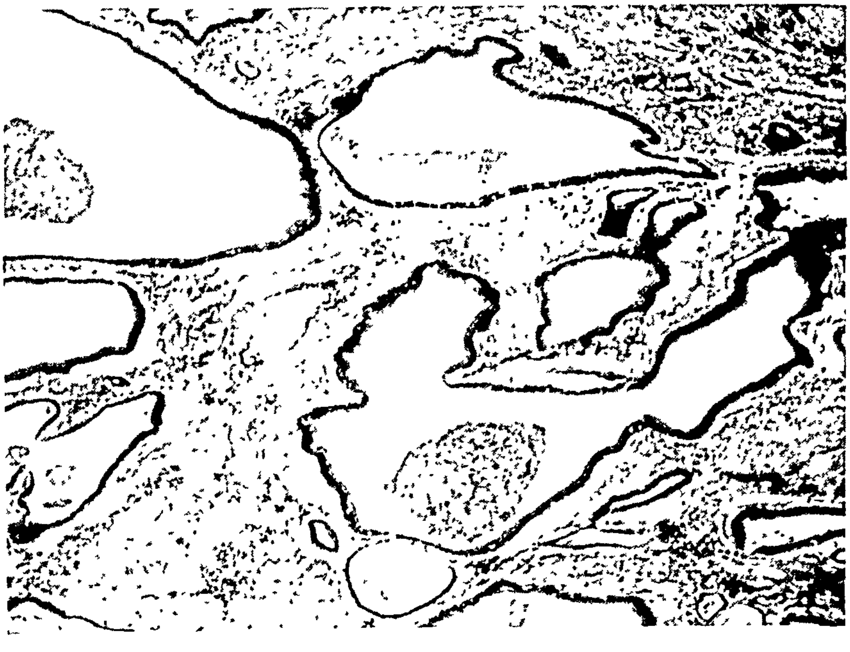

Fig. 2.—Photomicrograph of a section through a cystic portion of lung in Case 1 and showing numerous cystic structures variously lined by cuboidal, columnar, and ciliated columnar epithelium. Interstitial stroma is of loose connective tissue, and some cysts contain inspissated secretion. (Hematoxylin and eosin stain, ×200.)

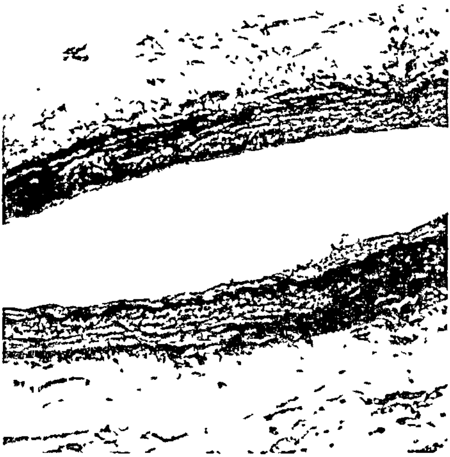

Fig 3.—Photomicrograph of a section through a third order pulmonary arterial branch in Case 1. Note early intimal thickening in lower right hand corner. (Verhoeff's elastic tissue stain, ×200.)

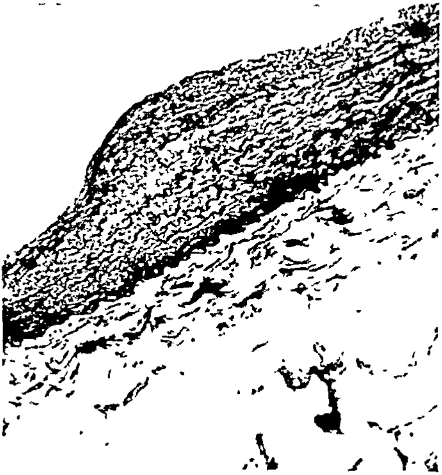

Fig 4.—Photomicrograph of a section through a primary artery to left lower lobe in Case 1. Note diffuse elastosis of intimal elastic plaque. (Verhoeff's elastic tissue stain, ×200.)

between the cysts consisted of connective tissue, histiocytes, and lymphocytes which frequently occurred as definite lymph follicles. Sections from other portions of the lobe showed normal lung tissue, except for the vessels. The large pulmonary arteries supplying uninvolved portions of the lobe showed early intimal plaque formation. These consisted of small subintimal accumulations of connective tissue and numerous small elastic fibers (Figs. 3 and 4). The arterial wall showed the usual number of relatively short fairly thick elastic fibers. In the incinerated preparations, such vessels showed increased mineral content in the wall. There was an accentuation of mineral deposition in the intima in some pulmonary arteries. Smaller arteries and arterioles in the uninvolved parts of the lung showed no deviations from normal and the venous system was also not remarkable. In one section a small bronchial artery in the wall of a rather large bronchus showed mild diffuse intimal proliferation.

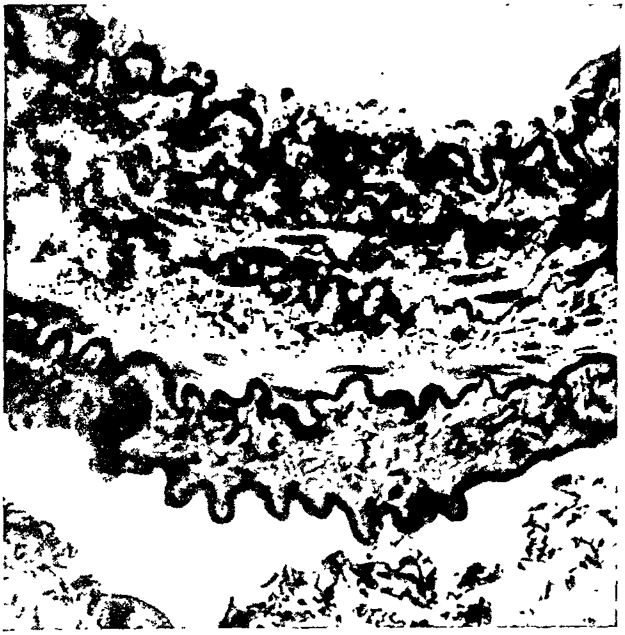

Fig. 5.—Photomicrograph of a section through a systemic artery in Case 2. This is characteristic of artery of hybrid type. Note greater proportion of muscle than in Figs. 4 and 8 and thicker, longer elastic fibers. (Verhoeff's elastic tissue stain, ×450.)

Sections through the systemic arteries showed them to have a different appearance from pulmonary arteries. These vessels were relatively much thicker walled than pulmonary arteries. Their media consisted of concentric lamellas of long undulating elastic fibers. In brief, these vessels had the appearance of hybrid arteries such as the visceral branches of the abdominal aorta (Fig. 5). They showed no evidence of intimal thickening or plaque formation and in incinerated preparations no increased mineral content could be seen (Fig. 6). In the posterior basal segment of the lobe numerous large arteries with the appearance of pulmonary arteries were found. Some of these large vessels were present even at the periphery of the segment in intimate association with the largest cyst. In many of these large pulmonary arteries very marked intimal thickening and plaque formation had occurred. In the micro-incinerated sections these vessels showed a considerably greater mineral content than comparably sized pulmonary arteries from uninvolved parts of the lobe (Fig. 7). Some of the sections showed in addition small branches of the aberrant vessels. These were readily identified by virtue of their different histologic structure and here, too, they showed no degenerative changes. Examination of the smaller, predominantly muscular branches of the pulmonary arteries showed changes in them which were as pronounced as those described in the larger arteries. There was extensive intimal thickening and some graying and splitting of the internal elastic lamella.

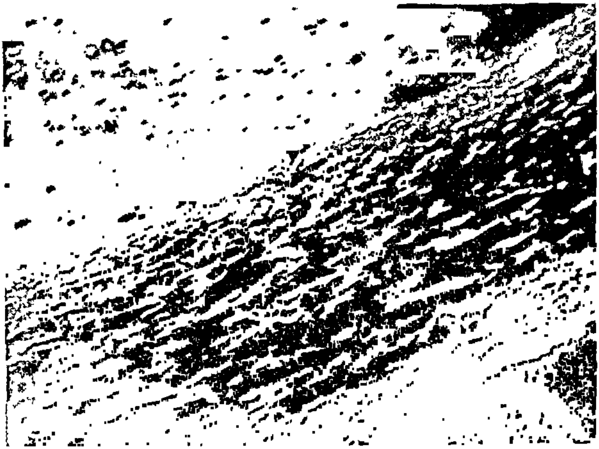

Fig. 6.—Photomicrograph of a micro-incinerated specimen Section through a major aberrant artery. Mineral ash is considerably less than in Fig. 7. White streaks represent nuclei of muscle cells of media (×150.)

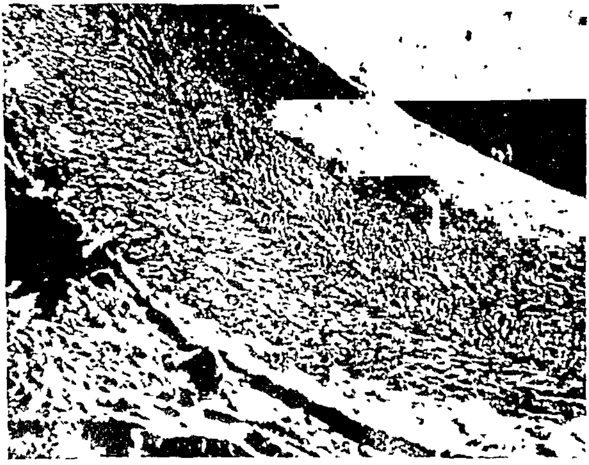

Fig 7—Photomicrograph of micro-incinerated section through a third order pulmonary artery branch in Case 1 Note white mineral ash (calcium) diffusely spread through the media A thin line of calcium at top of specimen represents endothelium, and clear area immediately underneath represents subendothelial fibrous thickening. (×450)

On micro-incineration some increased mineral content of the intima was again seen. These changes were not isolated and scattered, but rather involved the majority of the smaller vessels in all sections from the affected segment. The small and larger veins of this segment showed a very extensive phlebosclerosis. This consisted of marked intimal thickening and plaque formation and increased mineral content of the intima. The venous system in the uninvolved lung segments was normal.

In summary, the vascular findings consisted of isolated plaques in the large pulmonary arteries supplying normal segments of the lobe, and marked plaque formation and intimal thickening in the large pulmonary arteries in the affected segment. The smaller arteries in this segment almost universally showed marked intimal proliferation and the veins showed moderately advanced phlebosclerosis. The aberrant arteries were readily distinguished from the pulmonary arteries by virtue of their appearance either as that of a miniature aorta or a hybrid artery. They showed no degenerative changes.

Case 2.—(R. D. L. No. 3739.) A 60-year-old white man was admitted to the Jewish Hospital on June 1, 1937, with the history that in September, 1936, he had noted a raw area on the left buccal mucosa. He was operated on elsewhere in October, 1936, for a carcinoma of the cheek with excision of the ulcer and "parts of bone." Because of recurrence of the lesion, he returned to his local hospital for a second operation in December, 1936, and two weeks later, in January, 1937, the patient again noted a recurrent ulcer. For two to three days prior to his admission to the Jewish Hospital he had been unable to eat solids. He had no pain and there was no marked weight loss. He had chewed tobacco for many years. He had no respiratory symptoms. Physical examination on admission showed a man who was not acutely ill. There was a large clean ulcer in the left cheek with induration extending toward the nose and induration and swelling of the upper lip almost to the left commissure. There was a large ulcerated area on the buccal mucosa. The patient was able to open his mouth only with great difficulty and then only to a very slight degree. There were several enlarged firm, left anterior cervical nodes. The chest was emphysematous. Breath sounds were within normal limits. There were no râles. The remainder of the examination was within normal limits. On June 3, 1937, under general anesthesia, a radical dissection of the face and neck was carried out. On June 8, 1937, the patient suddenly became markedly dyspneic and died. An autopsy was performed.

Gross Pathological Description of Lungs.—The pleural cavities were largely obliterated by dense fibrous adhesions. These were particularly tenacious in the area between the base of the left lung and the left hemidiaphragm. The right lower lobe showed a small area of consolidation but there were no abscesses or cavitations. In the left lower lobe a large irregular cavity lined by smooth brownish tissue was found. A series of cartilage plates were found in one portion of the wall of this cavity and there were also numerous pockets and recesses. However, no connection between the cavity and the bronchial tree could be demonstrated. In the removal of this lobe, two arteries which had the appearance of large intercostal arteries were encountered. They originated about 5 cm. above the diaphragm from the thoracic aorta and entered the posteromedial aspect of the left lower lobe. They communicated with a large artery which coursed along the posterior aspect of the cavity.

Microscopic Description.—For this study there were available blocks of tissue from the right lung and blocks from various portions of the left lower lobe. There were no blocks through the anomalous systemic arteries. Hematoxylin and eosin stains, Verhoeff elastic tissue stains, and micro-incinerated slides were studied. The sections from the right lower lobe showed the normal pulmonary architecture to be well preserved. There were patchy and confluent areas of bronchopneumonia but no other abnormalities. Sections from the left lower lobe showed only scattered areas in which the normal alveolar structure of the lung was recognizable. The grossly observed cyst consisted of an inner lining of cuboidal ciliated epithelium. This was surrounded by connective tissue in which smooth muscle bundles occurred. Irregular cartilage plates were present in the cyst wall and there were many bronchial mucus glands. The surviving pulmonary alveoli and small bronchioles and bronchi in the remainder of the lobe showed infiltration with lymphocytes, polymorphonuclear leukocytes, and macrophages. Intervening between these persisting islands of lung tissue was a dense connective tissue stroma in which other scattered pulmonary structures could be found. There were small cystic structures lined by cuboidal epithelium, irregular cartilage masses unassociated with bronchial structures, and masses of lymphoid and fatty tissue. In several sections from the wall of the large cyst, one could see masses of a tumor which was composed of small deeply staining rather regular cells with relatively large nuclei. These nuclei, while relatively homogeneous, did show some variation in appearance. Some were pyknotic and deeply staining, while others were rather vesicular. Mitotic figures were rare. The cells were arranged in small masses which had a pseudoacinar or alveolar appearance. These cell masses were separated from each other by delicate connective tissue septa. The tumor was invasive but had not ulcerated or invaded the overlying epithelium of the cyst.

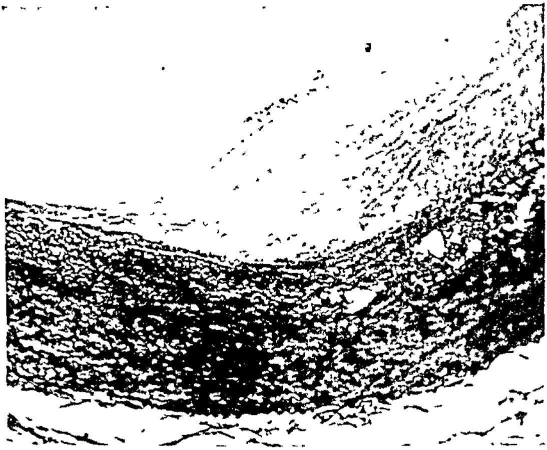

Fig. 8.—Photomicrograph of a section through primary artery to left lobe of Case 2 Note thick hyaline intimal plaque and dense elastosis of media beneath plaque. (Verhoeff's elastic tissue stain, ×200)

Study of the vasculature of the lungs resulted in the following observations. In the right lower lobe, the vessels were not unusual. Mild to moderate degrees of arteriosclerosis of the pulmonary arteries was present and consisted of intimal thickening and formation of scattered small plaques. The internal elastic membrane of many arteries was split or frayed. The veins were normal. In the left lower lobe, large pulmonary arteries occurred far out at the periphery of the lobe. They were in close association with the cyst. Their histologic appearance was characteristic of pulmonary arteries and they were the seat of a most advanced arteriosclerosis. Large subintimal connective tissue deposits and huge plaques of hyalinized connective tissue were common (Fig. 8). In many of these arteries the lumen was decreased by as much as one fourth to one third. The internal elastic lamella was thick, split, and hyalinized. In micro-incinerated sections these arteries showed a very marked increase in mineral ash content throughout their wall. Smaller arteries in the left lower lobe also showed marked degenerative changes. Intimal thicken-

ing and increased mineral content of the intima were again observed. At the same time these sections revealed an unusually intense phlebosclerosis. In some instances, this almost rivaled the severity of the arterial changes.

COMMENTS

These 2 cases emphasize several points which have not previously been reported. Perhaps one of the most important to the practicing surgeon is the presence of multiple aberrant systemic arteries in both of these cases, six in Case 1 and two in Case 2. From the surgical viewpoint, this means that every caution must be exercised in the dissection of such lobes even after one aberrant artery has been found and safely divided. The occurrence of a bronchial adenoma of the oncocyte-carcinoid type in the wall of the cyst of Case 2 is of equal importance. The malignant potentialities of these tumors are generally recognized today. Their not infrequent coexistence with congenital abnormalities of the lung is not so widely appreciated, but this is one of the important indications for surgical intervention for apparently benign cystic lesions even in the absence of clinical symptoms.

From our observations it would appear that, contrary to the view expressed by Pryce and others, the aberrant arteries are not pulmonary in type. Unfortunately, our second case was autopsied 15 years ago and we did not have sections through the arteries themselves available to us. However, in Case 1 these arteries differed strikingly from pulmonary arteries. The walls of aberrant vessels are thicker and the media contains long, plump elastic fibers arranged in concentric lamellas. These vessels also contain proportionately more muscle tissue than the pulmonary arteries. In the latter the elastic fibers are short and irregular, and the arteries are relatively thin walled. The aberrant vessels thus resemble systemic elastic or hybrid arteries. They are built to withstand high pressures. In Case 1, the aberrant vessels showed no degenerative stigmas. By comparison, the arteries and the veins in the affected lung segment showed advanced sclcrosis and even the larger pulmonary arteries supplying uninvolved segments showed early arteriosclerosis to a degree which one would not expect in so young a subject in the absence of pulmonary hypertension. Even in the older man in Case 2, the arteries and veins of the left lower lobe showed changes much more severe than one would expect solely on the basis of age and too such changes were absent in the vessels of the normal right lung. These changes in the pulmonary arteries are similar to those described by Gray and co-workers[16] in pulmonary hypertension due to mitral stenosis and other causes. Pareira and associates[17] have shown that elevation of pressure plays an important role in the development of arteriosclerotic lesions. This is particularly true in the case of pulmonary vessels which are not constructed to withstand the higher systemic blood pressure. These facts point to the presence of a systemic-pulmonary shunt.

As one would expect, the pulmonary vessels in the immediate vicinity of the aberrant systemic vessels sustain the greatest damage due to increased pressure. However, this hypertension is apparently reflected, although to a lesser degree, in all parts of the pulmonary circulation. The advanced phlebosclerosis is probably related to the presence of communications between the aberrant arteries and the pulmonary venous system. The presence of such communications is suggested by the injection studies of Lemmon and associates[18] and also by our own study of a recent case of bronchopulmonary sequestration. In this case injection of the aberrant artery with Lipiodol, a material which because of its viscosity would hardly be expected to fill rapidly vessels of capillary size, resulted in almost immediate visualization of the pulmonary vein (Fig. 9). Taking into consideration the structure of the aberrant arteries, it is not surprising that they show the least evidence of degenerative change. It seems logical to assume, however, that with progressive pulmonary arteriosclerosis and phlebosclerosis, the pressure in the aberrant artery would eventually increase so that it, too, would finally show arteriosclerotic stigmas. Previous authors noted the occurrence of such changes in aberrant arteries, but they failed to study the degree to which the pulmonary vessels shared in this process. We believe that their cases represent the end result of a process of which our Case 1 presents an early stage.

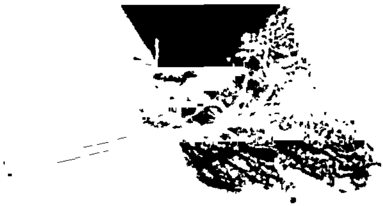

Fig. 9.—Radiograph of a left lower lobe with bronchopulmonary sequestration after injection of aberrant artery with Lipiodol. Cannula is in artery. Arrow points to pulmonary vein.

SUMMARY AND CONCLUSIONS

Two cases of congenital cystic malformation of the lung with multiple aberrant systemic arteries are presented. The importance of guarding against injury to these vessels in the course of lobectomy has been emphasized. In particular, one must continue to be on the lookout for further aberrant vessels if one such artery has been found. In one of our cases, a bronchial adenoma was superimposed on the congenital abnormality, thus underscoring once more the need for removal of all undiagnosed lesions of the lung.

Histologic study of the intra- and extrapulmonary portions of the aberrant arteries suggests strongly that they are systemic in origin. It was found that the aberrant systemic arteries bring about arteriosclerotic changes in the

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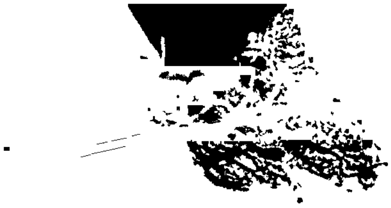

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PLASMA CELL GRANULOMA OF A MEDIASTINAL LYMPH NODE WITH EXTENSION TO RIGHT LUNG

M. A. Spyker, M.D., and S. Kay, M.D.
Richmond, Va.

PLASMA cell neoplasms and plasma cell granulomas often cannot be separated by any satisfactory criteria. Both behave in a similar manner, showing focal and/or diffuse infiltration with complete or incomplete obliteration of the normal architecture. If the destruction is complete, a neoplastic process is often inferred, and if incomplete, it may be either process. In general, these lesions have been given neoplastic significance when encountered in the lung. Analysis of these cases shows that they may just as reasonably be granulomatous. None of the patients in the reported cases has died due to metastases.[1, 2, 3, 6, 7] Some of the extramedullary plasma cell tumors in other sites caused the death of the patient.[5] Multiple myeloma may metastasize to soft tissue.[4, 8] The following case is an example of a mediastinal lymph node infiltrated by plasma cells with extension into the adjacent pulmonary parenchyma.

CASE REPORT

Mrs. D. M. G., a 37-year-old white married woman, was admitted to the Sheltering Arms Hospital in Richmond, Va., in February, 1954. Her chief complaint concerned a density in her chest that was discovered in a routine roentgenogram, in 1942, and which was diagnosed as a tuberculous lesion. The examination had been prompted by an increasingly severe low back pain, which had been present for several years. The patient was then sent to Blue Ridge Sanitarium, from where, after six weeks of diagnostic study in which tests were negative for tuberculosis, she was discharged to her home. She had had repeated roentgen examinations at six-month intervals since her discharge in 1942, up to the present admission. She had never had symptoms referable to the lesion, other than backache. She was told in the ensuing years that the lesion as seen on the original film had not changed materially. The review of symptoms was essentially negative. A history of operations revealed that she had had a thyroidectomy in October, 1943, for thyrotoxicosis. The family and occupational history were noncontributory.

The physical examination revealed a well-developed, well-nourished woman in no apparent acute distress. She weighed 118 pounds. On admission, her temperature was 99.8° F., blood pressure 160/58, pulse 84, and respirations 24. The eye, ear, nose, and throat examinations were essentially negative. The anterior neck region contained an old, well-healed, crescent-shaped scar. The thyroid was not felt and the trachea was in the midline. The lungs expanded well and equally and were clear to percussion and auscultation. No râles or wheezes were heard. The heart sounds were of good quality, without murmurs or thrills and with a regular rate. The abdomen was flat, soft, and nontender. No masses were felt. The pelvic and rectal examinations were negative. The urine was normal except for a trace of albumin. The blood count was essentially within normal limits. One determination of plasma proteins showed albumin 4.4 Gm. and globulin 2.6 Gm. The Kline test was negative.

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Roentgenograms of the chest revealed a density in the right hilus with an increase in multiple densities of the entire right lung field. The left lung field was not involved. On comparing the 1942 films with subsequent films, no prominent change was noted from that seen on the original examination.

The bronchoscopy before surgery revealed a normal larynx, moderate blunting and widening of the carina, and no visible tumor or stenosis.

An exploratory thoracotomy was performed on the sixth hospital day. The right lung was removed along with a mediastinal tumor that involved almost all of the upper and middle lobes and a portion of the lower lobe. The postoperative course was uneventful and the patient was discharged on the twenty-third hospital day.

PATHOLOGIC FINDINGS

Gross Examination.—The specimen consisted of the entire right lung measuring 17 by 13 by 3 cm. The organ was about normal in size, shape, and weight. The pleura was generally granular and fibrous, except for the part over the lower lobe. In the upper half of the hilar region there was a crescent-shaped hollowed-out area surrounded by compressed lung tissue. This defect had been originally filled with tumor. There were many pieces of tissue received separately to make a mediastinal tumor mass measuring 4 cm. in size altogether. It was friable, white, solid tissue. There was a slightly thickened rim of fibrous tissue around the defect in the lung. The lung parenchyma showed thickened nodular areas throughout. These were fibrous and crepitation was minimal. The bronchi were patent and apparently normal except for slight thickening of the walls adjacent to the tumor. The pulmonary arteries and veins were grossly normal. The bronchial lymph nodes were enlarged, firm, and black. Several 0.5 mm. subpleural black, firm nodules were present.

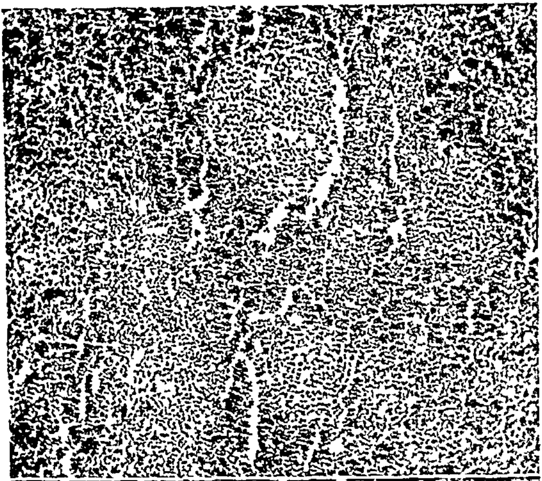
Microscopic Examination.—Section of the mediastinal mass showed a lymph node largely replaced by plasma cells (Figs. 1 and 2). These cells varied slightly in size and shape. The nucleus was large and hyperchromatic in a few cells. Russell bodies were prominently seen throughout (Fig. 2). The medium-sized and small blood vessels showed a varying amount of intimal fibrosis with encroachment upon the lumen causing almost complete obliteration (Fig. 3). The pulmonary tissue was replaced by focal and diffuse plasma cell infiltration and associated with hyalinization of fibrous tissue. Sometimes eosinophilic hyaline bodies were found replacing normal pulmonary parenchyma (Figs. 4 and 5). These hyaline bodies consisted of broad eosinophilic bands. The bronchial mucosa and submucosa were infiltrated by plasma cells, but the cartilage was not invaded by the cellular infiltrate (Fig. 6).

Pathologic Diagnosis.—Plasma cell granuloma of a mediastinal lymph node with extension to right lung.

DISCUSSION

The published cases concerning these plasma cell lesions are presented in Table I. The pathology was usually found on roentgen examination of the chest. Tuberculosis was eliminated by laboratory examination of the sputum. The usual laboratory tests were used to exclude multiple myeloma, and all were negative. Kuley and Kuntman[7] found 4 per cent plasma cells in the bone marrow, but attached no significance to this finding. Russell bodies were found in slightly less than one-half of the cases. In general, the clinical course of these cases had not progressed in accordance with recurrent or metastasizing neoplasms. We feel that, due to the long duration of the illness in our case, a granulomatous process is favored over the neoplastic. It may be that, at some future date, this patient will show evidence of multiple myeloma.

Fig 1



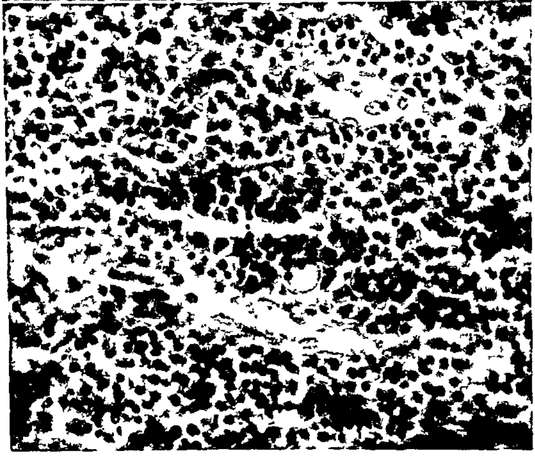
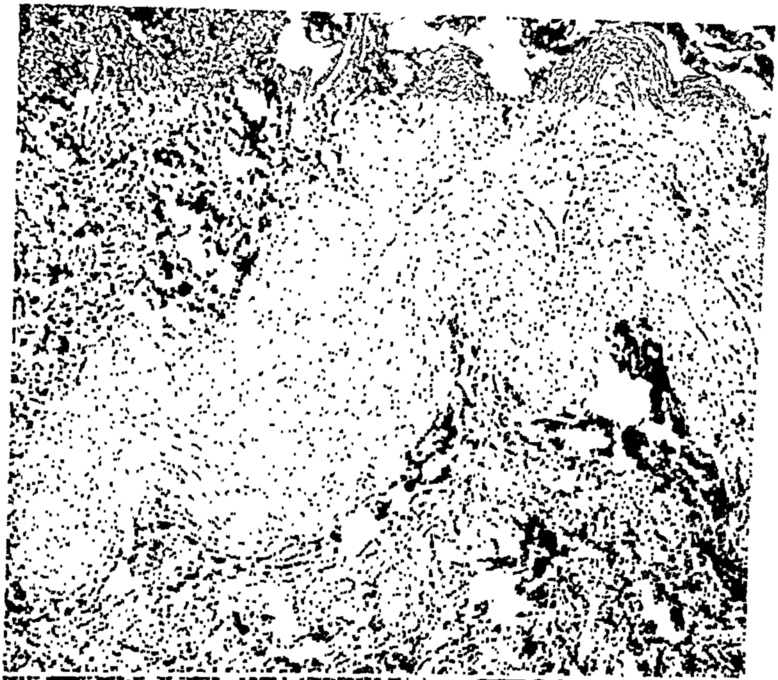


Fig 2

Fig 1 —Mediastinal lymph node low power

Fig 2 —Mediastinal lymph node, high power Most cells are plasma cells and a Russell body is in center of the field

Fig. 3.



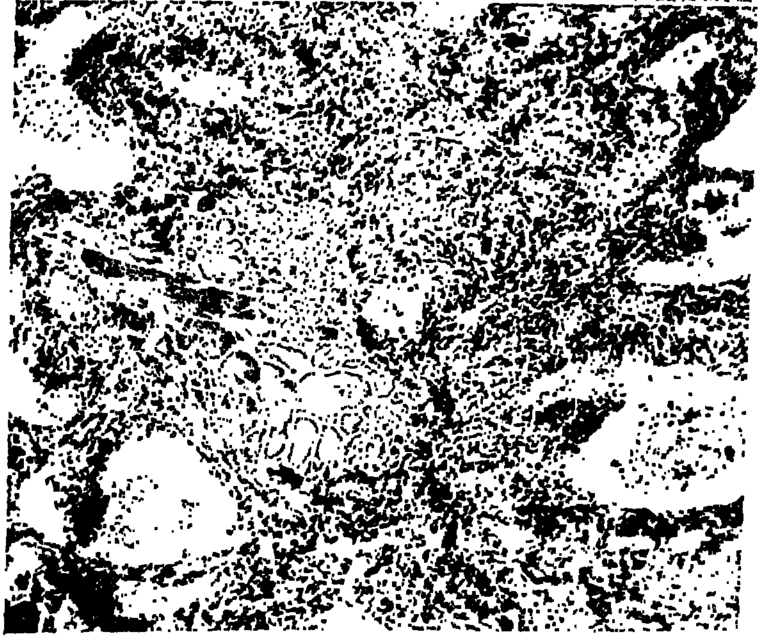
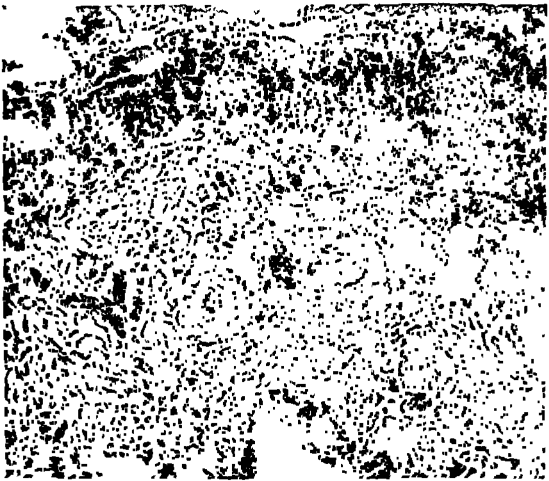


Fig. 4.

Fig. 3.—Lung, low power. Sclerosis of a large vessel due to marked intimal thickening.
Fig. 4.—Lung, low power. Granuloma associated with hyaline bodies.

Fig. 5.



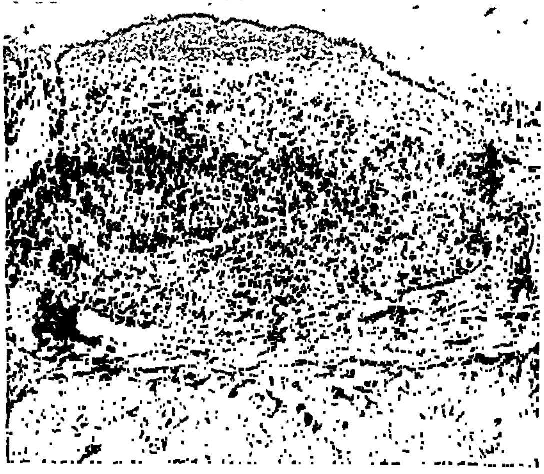


Fig. 6.

Fig. 5.—Lung, high power. Severe hyalinization of parenchyma.

Fig. 6.—Lung, high power. Diffuse sheets of plasma cells in wall of bronchus. The cartilage is not invaded.

TABLE I. REVIEW OF CASES IN THE LITERATURE

AUTHOR	AGE	SEX	ROENTGENOGRAM	PATHOLOGIC FINDINGS	AUTHOR'S DIAGNOSIS
Childress, and Addie[1]	37	M	Mass in left upper posterior mediastinum	6.5 cm. mass made up of plasma cells	Plasma cell tumor of pleura
	21	M	Mass in right middle lobe of lung	4 cm. mass containing plasma cells and Russell bodies	Plasma cell tumor of lung
Cotton, and Penido[2]	57	F	Mass in left upper lobe of lung	5 cm. mass infiltrated with plasma cells	Fibrosing plasmacytic granuloma of lung
Gordon, and Walker[3]	30	F	5 cm. mass in left upper inner lung field	5 cm. mass composed of plasma cells with Russell bodies	Plasmacytoma of lung, site of origin not determined
Hill, and White[6]	3	F	Mass filling left chest	8 cm. mass composed of plasma cells	Plasmacytoma of lung
Kuley, and Kuntman[7]	30	F	Mass in right hilus	Aspirate of mass contained 70 per cent plasma cells	Plasmacytoma of lung, site of origin not determined
Spyker, and Kay	37	F	Mass in right hilus with increased pulmonary markings	4 cm. mass containing plasma cells and Russell bodies with extension to lung	Plasma cell granuloma of mediastinum with extension to lung

There has been no roentgen or laboratory evidence of myeloma at the time of this writing. The presence of Russell bodies seems to support the contention that this is an instance of granuloma. We believe that the other six cases reported in the literature were apparently similar lesions and we agree with Dr. Fred Stewart[1-3] that most of these cases can be classified as granulomas.

SUMMARY

A case of plasma cell granuloma of the mediastinum and adjacent pulmonary tissue is reported. The lesion was present at least twelve years prior to its removal.

We desire to acknowledge Dr. W. A. Johns' permission to report his case from the Department of Surgery, Sheltering Arms Hospital, Richmond, Va.

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ARCHITECTURAL RECONSTRUCTION OF THE LUNG AFTER PARTIAL RESECTION OF PULMONARY PARENCHYMA

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COMPLETE re-expansion of the residual pulmonary parenchyma after partial resection is an essential condition for quick cicatrization, good functional recovery, and healing without the complication of empyema or bronchopleural fistula. Re-expansion depends on mechanical and anatomical factors.

An essential mechanical factor is the maintenance of patent bronchi which permits the air to reach the terminal bronchioles to ensure the ventilation of the alveoli so that maximum re-expansion of the lung may occur. To maintain re-expansion, pleural fluid must be aspirated by constant active suction.

The quality of the pulmonary parenchyma is an anatomical factor which facilitates or hinders the re-expansion. Normal pulmonary tissue is elastic and able to be distended by intra-alveolar pressure. As a result, it will usually replace the loss of substance caused by the operation. Yet, pathological types of parenchyma are frequent. The emphysematous lung, although relatively inelastic, may be distended to fill some of the remaining intrathoracic space. The fibrous lung, however, fixed by its rigid framework, has lost its elasticity, making hyperexpansion impossible. Finally the atonic lung, its texture being a mixture of fibrosis and emphysematous lesions, has no tendency to dilatation and will not occupy a space greater than it originally did. These varieties of lungs have, in other words, no tendency to play the spatial role described by Churchill, as essential in the refilling of the hemithorax after partial resection.

The analysis of the mechanism by which pulmonary tissue re-expands is based upon facts of current observation. First, the study of spontaneous healing of segmental lesions in tuberculosis shows that cicatrization is obtained by retraction. The diseased segment retracts, pulling and expanding the surrounding healthy parenchyma. The latter opens like a fan around its bronchial tree, while the diseased segment closes. This is the so-called Chamberlain's segmental clock. Furthermore, by observing the behavior of the hemithorax in children after partial resection, one is amazed at the facility with which the soft and resilient remaining parenchyma fills the thorax. Finally, the comparison between the postoperative courses of upper and lower lobectomies demonstrates the fact that the filling of the dead space poses no problem in the latter, whereas, in the former, difficulties are very often encountered.

Thus, one comes to the conclusion that three different factors play roles in the refilling of the dead space after partial resection:

1. Hyperexpansion of the residual parenchyma, possible because of the elasticity of the tissues.

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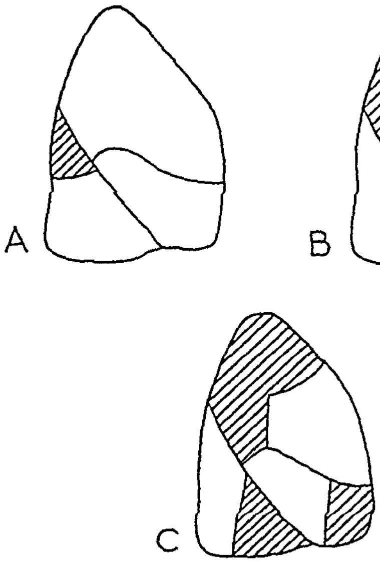


Fig. 1.—Types of resections where difficulties in refilling are encountered:
A, Resection of the superior segment of the lower lobe.
B, Resection of a large apical and posterior segment.
C, Resection of several segments.

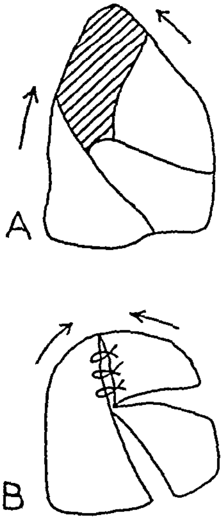


Fig. 2.—Fixation of the intersegmental resection plane of the anterior segment to the superior segment of the lower lobe (resection of the apical and posterior segments). Freeing of the fissures.

2 Elevation of the diaphragm, which depending on its integrity, diminishes the volume of the hemithorax

3 Displacement of the mediastinum whose mobility and whose ability to shift contribute also to the filling of the residual space

Observation also shows that (1) it is easier for the organism to obliterate a dead space by shift of a mobile organ diaphragm or mediastinum, than by hyperexpansion of the parenchyma except when the pulmonary tissue is healthy and elastic, which is rare in the adult and (2) when at the end of the operation, there remains a rigid parenchyma from which a pyramid shaped portion has been removed the refilling remains a difficult problem This is particularly true in isolated resection of the superior segment of the lower lobe and apical posterior segment of the upper lobe To avoid these instances, many thoracic surgeons perform thoracoplasty or plombage either in one operative procedure with the resection or, when a residual pleural pocket is evident, as a delayed postresection thoracoplasty Yet one must consider that this method complicates the operation, the postoperative course and causes skeletal deformities

These different facts led us to develop a method of architectural reconstruction of the lung which permits satisfactory re expansion of the lung at the end of the operation and avoids the postresection thoracoplasty

We started suturing edge to edge the raw intersegmental surfaces and then progressively elaborated a technique the basic principle of which is to bring the residual parenchyma in contact with the rigid parts of the thoracic cavity the apex of the hemithorax and costal wall Thus, the lung might be well separated from mediastinum and diaphragm These mobile structures are able to shift and fill the residual space

From this basic principle two necessary objectives have been derived

1 The reconstruction of a pulmonary pyramid proper to be reintegrated into the bony dome of the hemithorax We had observed that after certain partial resections of the upper lobe, after left upper lobectomy and after right upper and middle lobectomy, important pleural pockets remained in the apex Even good parenchymal expansion could not obliterate them Therefore, it seemed to us indispensable to reconstruct a pulmonary apex adaptable to the dome of the hemithorax

2 The achievement of a close contact between the pulmonary parenchyma and the bony structures of the thorax, costal wall, and vertebral gutter was a logical deduction from the previously mentioned facts and to us an imperative condition for the success of the operation It should assure complete re expansion and a proportional recuperation

To apply these two theoretical principles, we use certain technical procedures which will now be described

1 The lung is completely freed from its parietal fixation (unless there remains only a small retracted lower lobe) and, if necessary, its decortication Thus, the re expansion will be well equilibrated, and will involve all segments which will decrease tension on the areas adjoining the operative zone

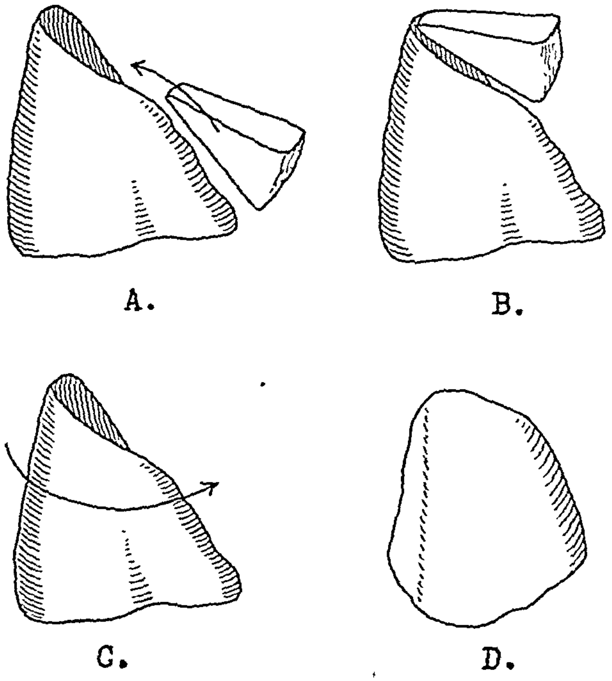


Fig. 3.—Rotation of a lobe.

A, After upper lobectomy the fissure between the middle and lower lobes is opened.

B, The posterior rim of the middle lobe is brought in contact with the apex of the superior segment of the lower lobe.

C and *D*, By rotation around its hilar axis the lower lobe is placed so that the posterior surface becomes lateral.

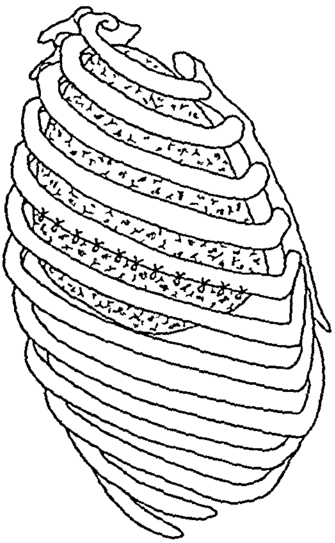


Fig. 4.—Once turned around its axis the lower lobe is pushed upward so that it will fill the apex of the hemithorax. It is maintained in this new position by a few fixation stitches to the chest wall.

2. The opening or the completion of the interlobar fissures has the same objective as the freeing of the lung, an equal distribution of the tension each lobe has to support. It will allow the bronchial tree to open and to distribute to each of its elements the corrective role of refilling the dead space.

3. The rotation of a segment or a lobe around its bronchial axis brings it in the required new position. This rotation must be done smoothly, avoiding, naturally, any kinking of blood vessels, allowing the vascular system to preserve its functional integrity. Surprising as it may seem, this rotation is in reality only a slight exaggeration of what nature does in its slow and spontaneous process of retraction when a lobe becomes atelectatic or carnified, the adjoining parenchyma replacing the loss of substance. It is also the process which occurs spontaneously to compensate for loss of pulmonary tissue after partial resection. We have verified this by bronchography in two collateral series of cases

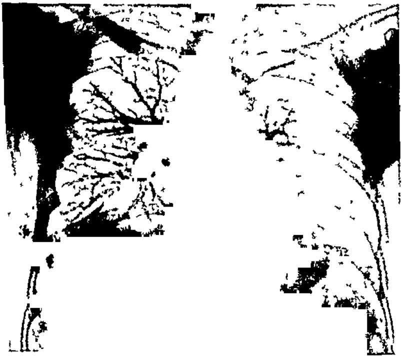


Fig 5

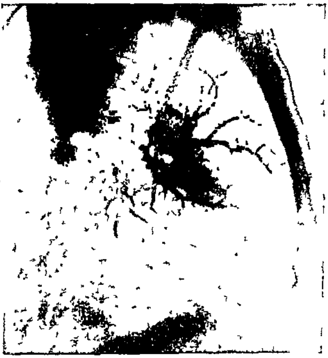


Fig. 6.

Fig. 5 —Bronchogram after right upper lobectomy.

Fig. 6 —After rotation and upward displacement, the middle lobe, hence, constitutes the apex of the lung. The bronchial tree has unfolded.

4. The fixation of the rotated lobe or segment in this new position is a complementary procedure to maintain the acquired position. This is done by a series of stitches fixing the raw intersegmental or interlobar surfaces together. The stability obtained by this architectural reconstruction shifts the hyperexpansion away from the operative area.

If necessary, we conclude the endothoracic procedure by securing the lung to the chest wall with a few stitches. A close contact will be assured between lung and chest wall even if a dead space has to be left near the mediastinum or diaphragm. We mentioned before that it seemed very important to us to fill the apex of the thoracic cavity with parenchyma and this is easily possible if the lung, remodeled in the form of a cone, is pulled upward. This procedure is indispensable in the resection of part of or the whole upper lobe and a suspension by suturing is always necessary here.

5. All of these manipulations must be done with the lung well inflated. Only the pressure given by the anesthetist's balloon, transmitted to the alveoli, allows one to judge how to place lobes and segments and assign them—the optimum definitive place.

This theoretical discussion, abstracting the different technical points, can be applied to almost all cases. Yet it seems useful to develop with more detail certain particular instances.

There follow three examples:

1. After right upper lobectomy or resection of the apical-posterior and anterior segments in the left, the lower, and middle lobes or the lingula, there is difficulty with expansion to fill the dead space in the adult. To re-create an apex, we open completely the fissure between the lower and middle lobes. The middle lobe or lingula is then drawn backward and upward until its posterior angle reaches the top of the superior segment of the lower lobe. This requires rotation around its hilar axis. The middle lobe is fixed with a few stitches to the lower lobe in this new position and will henceforth constitute the pulmonary apex.

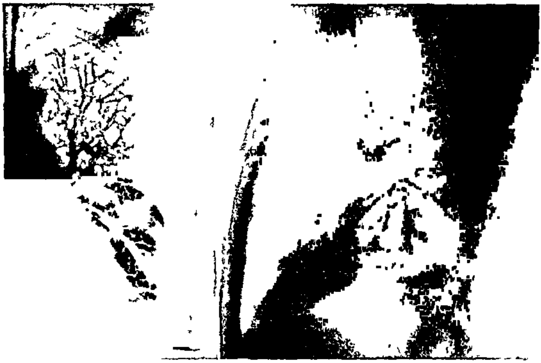
2. After resection of the apical and posterior segments, or the apical-posterior segment, we often free the great fissure between the right lower and middle lobes or left lower and upper lobes. We then slide the remaining parenchyma of the upper lobe backward until the raw resection plane comes in contact with the posterior edge of the lower lobe. As after upper lobectomy, a few stitches secure the lobes in their new position.

3. After right upper and middle lobectomy, or total left upper lobectomy, we section the triangular ligament which fixes the lower lobe and turn the latter around its hilar axis so that its posterior surface becomes lateral. The lobe is then pushed upward and is fixed to the thoracic wall. It fills the dome of the hemithorax. This suspension leaves the lower lobe distant from the diaphragm and mediastinum especially when it is a small-sized lobe.

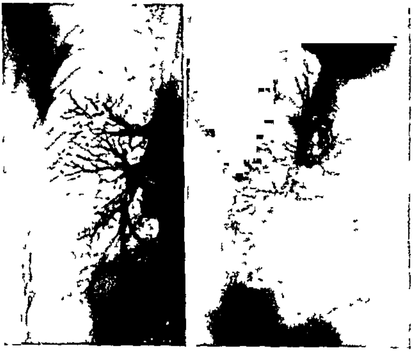
The results we have obtained with this method have always been good even in cases of fibrotic lungs. It is evident that this technique does not change the usual postoperative care that is required. An atelectasis might well tear the lung loose from its fixations. The postoperative surveillance must be as constant as in routine procedures and any bronchial obstruction or air accumulation in the hemithorax avoided or corrected immediately. We have operated thus on 136 patients during the last twenty months; 14 resections of a single segment, 58 resections of two or more segments, and 64 lobectomies for tuberculosis, bronchiectasis, residual abscesses, cysts, etc.

We have observed residual pockets following atelectasis in seven instances. Among these we noted four cases of localized empyema, three of which were easily cured, the fourth required further surgery. The three remaining cases, complicated with atelectasis, produced only simple air pockets. If it is difficult [illegible] hold our method responsible for these complications, one can nevert[illegible] gue that it did not prevent them.

Bronchograms with Lipiodol were made in 30 of our patients between the twelfth and twenty-first postoperative days to visualize the changes of the bronchial system after the structural modifications to which it had been subjected. These radiologic checkups never revealed any bronchial kinking. Some showed incurvations similar to those observed after spontaneous retraction in tuberculosis; others were in all points comparable to the bronchograms studied by J. Antrim Crellin, J. Stauffer Lehman, Daniel Mason, and J. L. Curry after partial lung resection. We observed mostly, on bronchograms made after lobectomy, a harmonious fanning out of the bronchial tree into its new area.



Figs 7. and 8.—After left lower lobectomy, the upper lobe has rotated slightly The lingula occupies now the lateral part of the hemithorax. Note the incurvation of several small bronchi



Figs 9. and 10—After resection of the anterior segment the apical segment has been fixed to the middle lobe by a few stitches The middle lobe bronchus is slightly curved

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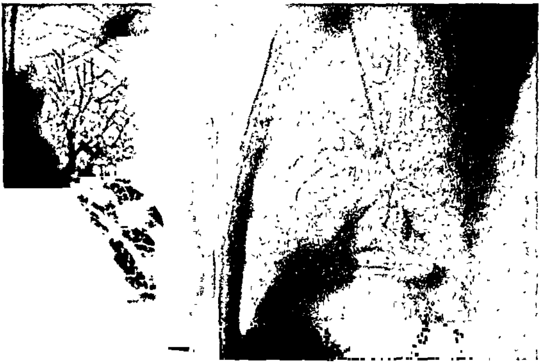
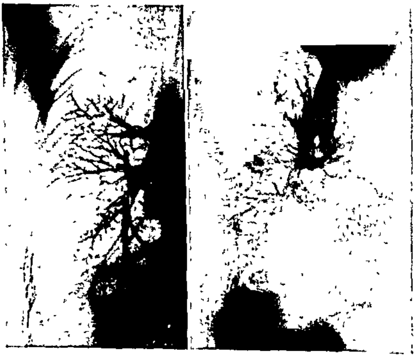
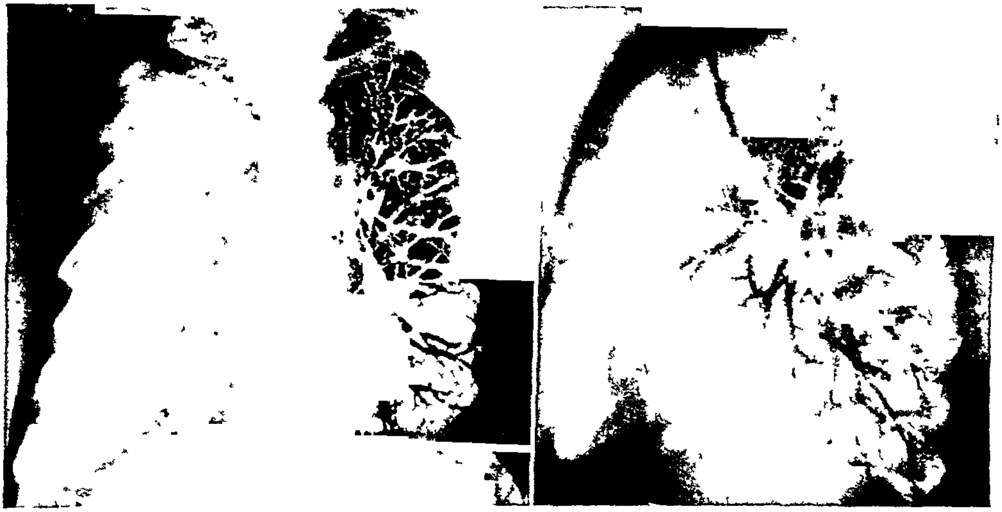


Figs. 7. and 8.—After left lower lobectomy, the upper lobe has rotated slightly. The lingula occupies now the lateral part of the hemithorax. Note the incurvation of several small bronchi.



Figs. 9. and 10.—After resection of the anterior segment the apical segment has been fixed to the middle lobe by a few stitches. The middle lobe bronchus is slightly curved.

One might question theoretically, whether the artificial reconstruction of the parenchyma is justified and whether one is justified in modifying a natural architecture, to impose new forms and positions to segments or lobes without risk of functional incapacitation. One can object that the fixation of the segments to one another, the fixation of the lung to the skeleton, in order to provide enlargement might interfere with the physiologic role of the lung and disturb the respiratory function. Up to the present, we have not made any detailed functional evaluations which might justify our technique, but



Figs. 11. and 12.—Resection of the apico-posterior and anterior segments. The lingula is now in the apex.

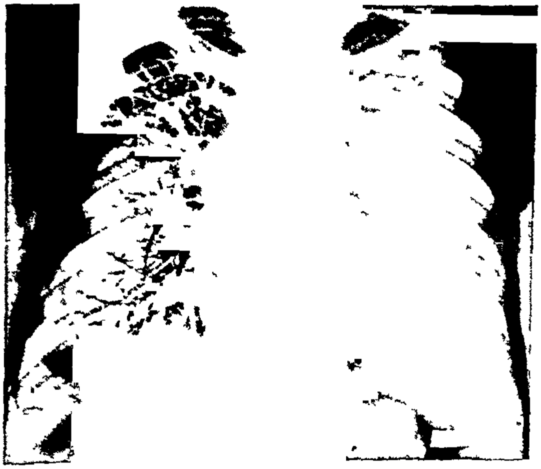


Fig. 13.—Resection of the apical and posterior segments of the right upper lobe. The fissure between upper middle and inferior lobe has been opened.

the excellent clinical results obtained, the normal postoperative films, and the radioscopic study which reveals normal *aeration*, have encouraged us to continue with our method. The fixation of the lung to the thoracic wall seems therefore not to be a serious handicap. The parietal adhesion of the lung is an inevitable consequence of every thoracotomy. The free sliding motion of the lung within the thorax, normal movement of any free parenchyma, disappears after all endothoracic operations and we do not think that the fixation to the chest wall with a few stitches changes anything. As to the change in the positions of segments or lobes, the slight incurvations of bronchi still allow perfect air circulation and the fact that they exist cannot be considered the consequence of the method alone.

The air leaks which one might expect to increase, because of the fixation stitches which damage the lung, have never been more significant than after routine procedures. They always stay within normal limits. The pleural pockets left near the diaphragm or mediastinum have always been obliterated in forty-eight hours by the molding of these mobile organs to the re-expanded residual parenchyma.

It is evident that this technique can be applied only when the remaining parenchyma is sufficient to refill the hemithorax. It is of no help if the residual lung is too small or insufficient. Two residual segments cannot refill a hemithorax in an adult.

Nevertheless, in many cases, the method has allowed us to refill a thoracic cavity perfectly; this could otherwise not have been achieved. We think that the architectural reconstruction of the lung is of great practical help and is theoretically justified.

SUMMARY

1. The re-expansion of the lung after partial resection is essential for the success of the operation.

2. To avoid postoperative complications and assure functional recuperation, this re-expansion must be complete. This depends on mechanical and anatomical factors.

Three mechanisms play roles in the refilling of the hemithorax: hyperexpansion of the remaining parenchyma, elevation of the diaphragm, and shift of the mediastinum.

4. Observation shows that there is no difficulty if after the operation the remaining lung is in contact with the rigid elements of the thoracic cavity (ribs and vertebral gutter) and distant from the mobile structures (diaphragm and mediastinum).

5. To ensure the refilling of the hemithorax, the basic principle of our technique is to bring the lung in close contact with these bony structures, leaving it distant from the mediastinum and diaphragm.

6. This technique requires a few procedures which allow an architectural reconstruction of the pulmonary parenchyma to a cone-shaped entity: complete freeing of the lung, freeing of the fissures, rotation of a segment or a lobe, fixation by stitches of segments and lobes one to another and, if necessary, fixation of the lung to the chest wall.

7. This method was used in 136 patients in lobectomies and segmental resections for various indications.

8. Postoperative bronchograms never showed bronchial kinking but only wide incurvations.

9. There seem to be no theoretical objections to this method.

EOSINOPHILIC GRANULOMA OF THE LUNG

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Duarte, Calif.

INTRODUCTION

Eosinophilic granuloma is a rare disease of unknown etiology which affects a multiplicity of tissues. While usually generalized, solitary involvement of bone,[52, 64, 76] of skin,[27] of the gastrointestinal tract,[12, 89, 100] and occasionally of other tissues[68] has been recognized. It has only recently been appreciated, however, that solitary involvement of the lung may also occur, and only two such reports can be found in the literature.[30, 67] It seems, therefore, appropriate to report a case recently encountered by us of eosinophilic granuloma of the lung without other demonstrable systemic disease.

CASE REPORT

D. B. W. (X12011), a 15-year-old white boy, was admitted to the Diseases of the Chest Hospital of the City of Hope Medical Center on May 12, 1953, because of the findings by a Mobile Unit chest x-ray survey in December, 1952, of extensive infiltration throughout both lung fields (Fig. 1). He was completely asymptomatic, without cough, expectoration, or hemoptysis. There was no fever, night sweats, loss of weight, weakness, fatigue, or dyspnea, and no frequency of urination, bone or joint pains, or skin lesions. He was admitted with a presumptive diagnosis of bilateral advanced pulmonary tuberculosis.

Past history included measles, and an unexplained episode of fever associated with left hip pain at the age of five, which lasted intermittently for one year.

Physical examination revealed a well-nourished, well-developed white boy, 15 years of age who appeared to be in excellent health. The temperature was 98.6°; pulse, 84; respiration, 22; blood pressure 120/76; height, 5 feet 9½ inches; the weight was 156 pounds. The skin was clear and there was no cyanosis. The eyes showed no exophthalmos. The oral mucosa, tonsils, and pharynx were clear. There was no cervical, axillary, or inguinal lymphadenopathy. The chest was symmetrical and expansile. The lung fields were resonant. Slight generalized accentuation of breath sounds was noted. No râles were present. The heart was normal. The liver and spleen were not enlarged. Rectal examination revealed no abnormalities. No skeletal tenderness or deformity was observed, and neurological examination was normal.

Laboratory Findings.—Blood count: The red blood count was 4,600,000; hemoglobin, 14 Gm.; white blood count was 9,800; segmented polymorphonuclear leukocytes 69, lymphocytes 21, monocytes 4, eosinophils 6. *Urinalysis:* specific gravity 1.026; albumin negative; glucose negative; acetone negative; microscopic examination revealed a few hyaline casts. *Bone marrow examination:* No abnormalities. *Sedimentation rate:* 19 mm. per hour. *Blood chemistries:* Fasting blood sugar 90 mg. per cent; nonprotein nitrogen 30 mg. per cent;

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total protein 7.4 Gm.; albumin 4.8 Gm.; globulin 2.6 Gm.; calcium 11 mg. per cent; phosphorus 3.7 mg. per cent; cholesterol 140 mg. per cent; cholesterol esters 70 per cent; amylase 32 units; bromsulfonphthalein (5 mg./kg.) 8 per cent dye retention after 45 min.; thymol turbidity 3.5 units; alkaline phosphatase 5.9 units (Bodansky); icteric index 6 units; quantitative van den Bergh test less than 0.25 mg. *Thorn test:* 75 per cent drop in circulating eosinophils. *Bacteriology:* there was no sputum raised; nine gastric examinations were negative on culture for acid-fast bacilli. Culture and smear of pulmonary biopsy specimen (including special procedures for fungi) revealed no organisms. A Mazzini test was negative. *Coccidioides serology:* complement fixation 2 plus in 1:2 dilution, precipitin test was negative, findings were considered equivocal. *Skin tests:* the Mantoux test was 4 plus in 1:10,000 dilution; coccidioidin test was negative in 1:1,000 dilution; histoplasmin was negative. *Stool examination:* negative for fat, undigested muscle fiber, and starch granules. *Electrocardiogram:* normal. *Vital capacity:* 3,700 c.c.; 72 per cent of normal. *Breath holding time:* 72 seconds.

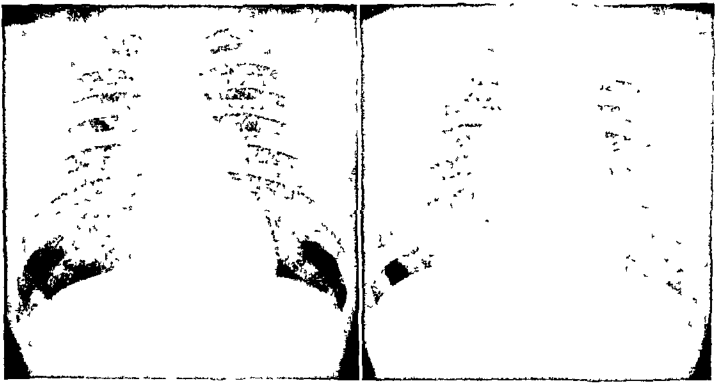

Fig 1. Fig. 2.

Fig. 1.—X-ray of chest shortly after discovery of the disease. Note diffuse soft nodular infiltration and suggestion of fibrocystic changes

Fig 2.—X-ray of chest prior to operation, showing some progression of the infiltrative process

Clinical Course.—Despite modified bed rest at home for two months, the admission film showed an apparent progression of the pathology throughout both lung fields (Fig. 2). There were heavy root shadows and scattered mottling by soft nodular densities and areas suggestive of multiple small cystic areas or cavities from the apex to the base bilaterally. The patient was placed on strict bed rest and an antibiotic regimen consisting of streptomycin 1 Gm. twice weekly and para-aminosalicylic acid (PAS), 12 Gm., and isonicotinic acid hydrazide (INH), 150 mg., daily. Repeated examinations of gastric contents were negative for acid-fast bacilli, and the patient remained completely afebrile and asymptomatic. Bronchoscopy was performed and revealed diffuse erythema of the mucosa, but was otherwise normal. Scalene fat pad and lymph node biopsies were performed and showed no evidence of nodular or granulomatous lesions.

In view of the failure to recover the tubercle organism, it was felt that exploratory thoracotomy and lung biopsy were indicated to establish a definite histologic diagnosis, and this was performed on Oct. 2, 1953. At operation through a right anterolateral approach through the fourth interspace, the pleural cavity was found to be free of adhesions or fluid. Small cystic areas containing air, the largest measuring approximately 1 cm. in diameter, were scattered over the surface of the lung; and on palpation, shottylike nodules from 0.3 to 0.7 cm. in diameter were felt scattered diffusely throughout the parenchyma. There was no hilar adenopathy. A biopsy specimen 3 cm. in width was taken in wedge fashion from the upper lobe, and a second similar specimen was removed from the middle lobe. Postoperative convalescence was completely uneventful.

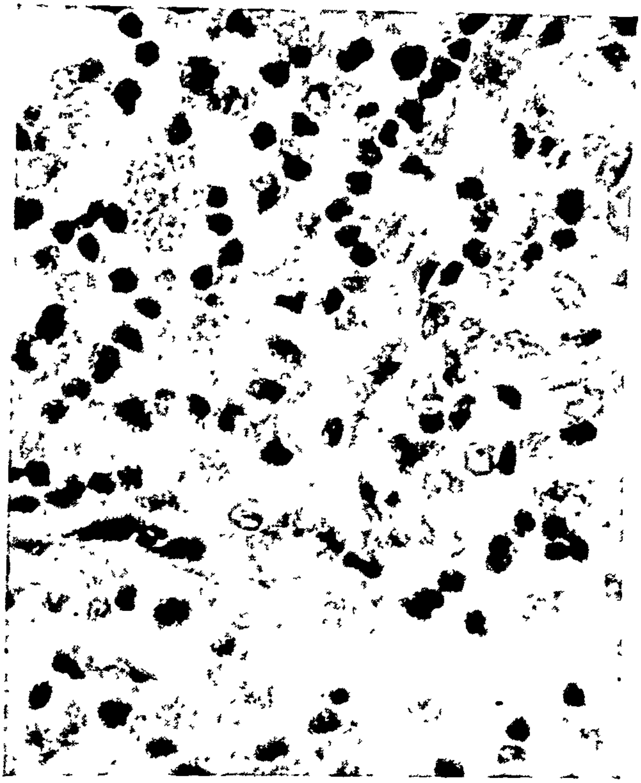

Fig. 3.—Photomicrograph of biopsy specimen, high power, showing histiocytic infiltration, profusion of eosinophiles, xanthomatous degeneration of histiocytes, phagocytosis of pigment granules by histiocytes, and fibroblastic proliferation.

Pathology Report.—Gross: The first biopsy specimen was a small wedge of peripheral lung parenchyma which included a dilated bronchial branch which led into an empty thin-walled multicystic area, and was lined by a glistening grayish-white surface a little thicker than the normal visceral pleura, with loculations up to 0.6 cm. in diameter. The second biopsy specimen was a small wedge of crepitant pink peripheral lung parenchyma containing several thin-walled empty glistening cystic areas, up to 0.4 cm. in diameter, without obvious bronchial connection. The walls of the cysts offered increased resistance to cutting despite their thinness. *Microscopic:* The histologic appearance of both specimens was similar (Figs. 3 and 4). The pleura was slightly thickened. Throughout the lung parenchyma there were aggregates of large fusiform or stellate-shaped histiocytes with vesicular nuclei, intermixed with abundant eosinophils. Occasional giant cells were noted. Small collections of brownish granular pigment were noted within some histiocytes. While in some places the aggregates of histiocytes, eosinophils, and giant cells were within the alveoli, for the most part they replaced normal lung parenchyma. Several of these nodular areas showed varying amounts of fibrosis, and in several places there was extensive fibrous replacement of the lung

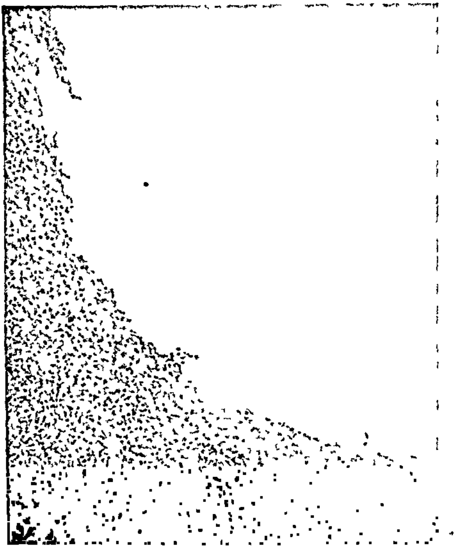

Fig 4.—Photomicrograph of biopsy specimen, lower power, showing cyst wall

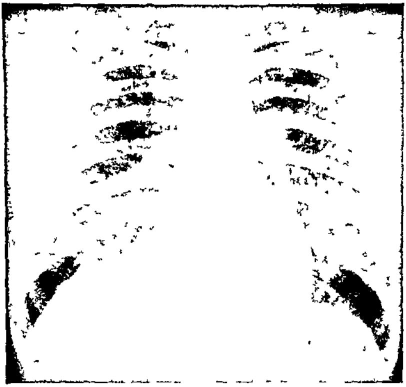

Fig 5—X-ray of chest nine months postoperatively and nineteen months after discovery of disease. There is slight clearing of the infiltrative process [illegible] right diaphragm has occurred

parenchyma, with only a few histiocytes or eosinophils recognizable. In places, the lung parenchyma showed emphysema or cystlike spaces lined by fibrous or granulomatous tissue. Occasional bronchiolar branches led into these cysts. Diagnosis: Eosinophilic granuloma, lung.

Subsequent course: Following establishment of the diagnosis, a complete bone survey was performed, but failed to disclose any abnormalities. Because of reports[5, 7] in which it was felt that cases of Letterer-Siwe's disease had been ameliorated by antibiotics, it was decided to treat this patient with a course of combined antibiotic therapy. He had already received streptomycin 1 Gm. twice weekly for three months, as well as PAS and INH. He was now given repository-type penicillin 600,000 units daily, streptomycin 1 Gm. daily, and Aureomycin 250 mg. 4 times daily for the next three months, following which he received erythromycin 250 mg. 4 times daily for the next two months. The last x-ray examination made of his chest, nine months postoperatively and nineteen months after the disease was first noted, revealed some slight decrease in the extent of the bilateral infiltration. Postoperative tenting of the right diaphragm from pleural adhesions was present. There was no evidence of a new process or of recent spread (Fig. 5). The patient was discharged to his home and was leading a normally active life twenty months after the disease was first noted. He was asymptomatic at that time, and continued to show no evidence of extrapulmonary involvement.

DISCUSSION

It is now convincingly established that the pathological basis of eosinophilic granuloma,[23, 33, 36, 64, 76] Hand-Schüller-Christian syndrome (lipogranulomatosis or normocholesteremic xanthomatosis),[13, 14, 17, 46, 54, 81, 82, 85, 86] and Letterer-Siwe's disease (reticuloendotheliosis or histiocytosis),[1, 61, 90] is fundamentally the same.[28, 29, 42, 44, 47, 49, 52, 53, 63, 64, 66, 94, 95, 96] Lichtenstein's paper[63] is particularly definitive on the subject. Pulmonary manifestations in these conditions have usually been recorded as incidental findings in a generalized disease process in which the diagnosis of the pulmonary lesion was either inferred from positive extrapulmonary tissue biopsy, or proved at autopsy.[1-4, 6, 8, 10, 11, 13-15, 18-23, 25, 26, 29, 31, 34, 35, 37-39, 41, 43-46, 48-52, 56-60, 62, 65, 68, 69, 71-75, 77-81, 83, 84, 87, 88, 90-93, 96-99, 102-104] In only a few cases have the pulmonary manifestations themselves been the focus of interest. Actual confirmation of the pulmonary pathology in the living subject by lung biopsy, in cases where extrapulmonary manifestations existed, has been reported only 6 times.[8, 35, 63, 66a]

Clinical recognition and pathological confirmation by lung biopsy of the existence of eosinophilic granuloma as an *isolated* disease entity of the lung, without other demonstrable systemic involvement, was first reported by Farinacci, Jeffrey, and Lackey, in 1951.[30] (Their 2 cases were re-reported by Lackey, Leaver, and Farinacci, in 1952,[58] and again referred to by Lichtenstein, in 1953,[63] and Forsee and Blake, in 1954.[35]) Mazzitello, in 1954,[67] reported 2 new cases. The present report is believed to be the fifth reported case of isolated pulmonary involvement. It must be assumed, not that the disease did not exist in primary pulmonic form in the past, but that in the absence of characteristic systemic lesions by which the pulmonary lesions could be recognized, the disease process in the lungs was incorrectly interpreted. As yet, to the best of our knowledge, there has been no report of a case starting out exclusively with pulmonary involvement and subsequently developing generalized disease. Eventual recognition of this missing link is to be anticipated.

CLINICAL FEATURES

Eosinophilic granuloma involves tissue either in the form of discrete nodular masses of granulomatous tissue of varying dimensions, or in the form of diffusely disseminated granulomatous tissue. In the larynx, fibrotic stricturing may occur following cicatrization of the granulomatous lesion therein. In the tracheobronchial tree, polypoid and papillary protrusions of granulomatous tissue have been described,[35, 81, 82] which may produce gross bronchial obstruction. Diffuse plaquelike involvement of the respiratory mucosa of the tracheobronchial tree has also been noted, and stricturing may occur at any point. Mediastinal and hilar lymphadenopathy may be extensive. The pleura is often found studded with granulomatous nodules, but may be involved in a diffuse plaquelike manner. The pulmonary parenchyma proper may be involved by a few large single or multiple nodules, but more often there is a profusion of nodules of smaller size, commonly ranging from miliary proportions to 2 cm. in diameter, giving the tissue a characteristic "shotty" feel. Diffuse granulomatous infiltration of the interstitial parenchyma is also usually present, and in later stages parenchymal fibrosis becomes advanced. Going hand in hand with these changes are the cystic changes which result from bronchiolar obstruction, produced by actual granulomatous obturation of bronchiolar lumina and by cicatricial constriction and stenosis of the lumina following interstitial fibrosis. Granulomatous degeneration of the alveolar wall may be an accessory factor in cyst formation. These cystic changes may range from a few small surface blebs, through increasing emphysematous degeneration, to complete cystic degeneration with giant bullae or honeycombed lungs in the end stages.

Microscopically, the pathologic features of the disease are classically subdivided into four phases on the basis of progressive age:[49]

1. In the hyperplastic-proliferative phase, there is accumulation of histiocytes, which appear in strands and sheetlike collections. Some are actively phagocytic and some are multinucleated. There is an associated inflammatory cell infiltration consisting primarily of eosinophilic polymorphonuclear leukocytes, but including plasma cells and round cells in lesser numbers.

2. In the granulomatous phase, there is increased growth of blood vessels and stroma in the area of histiocytic proliferation. Fibroblastic activity is noted, and reticular fibrils are laid down. The previously noted inflammatory cell infiltration composed primarily of eosinophils is conspicuous, and giant cells may be noted. Areas of hemorrhage and necrosis may be found.

3. In the xanthomatous phase, isolated histiocytes or nests of histiocytes undergo transformation into foam cells.

4. The fibrotic phase is the end phase, or that of healing. Cellularity of the pathologic picture diminishes as fibrosis increases. Fibrosis is noted to some extent in all stages, but eventually becomes quite pronounced.

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There is no strict demarcation between the different stages and much overlapping occurs. The same pathological specimen may show different pictures in different areas, and elements of all may be found in any one field.

The disease may be present at any age, and in both sexes. The patient with pulmonary involvement may be completely asymptomatic, as was the patient reported in this paper. On the other hand, he may complain of chronic productive cough, bloody streaking of the sputum, or at times frank hemoptysis. Low-grade fever may be present, more marked in the afternoon and asssociated with night sweats. Weakness, fatigability, and weight loss are constitutional concomitants. Chest pain may be present. Pleural effusion may occur. Spontaneous pneumothorax, frequently bilateral, may occur following the rupture of emphysematous blebs. Dyspnea of increasing severity may be present, and as the disease progresses respiratory decompensation may become extreme. In addition, progressive right heart failure may eventually result from the extensive pulmonary fibrosis.

In early stages, chest x-ray films may show a generalized reticulation associated with miliary mottling, and later there may be diffuse soft nodular infiltrations of varying dimensions. Large irregular densities may be seen. Fibrotic changes are a constant feature, and, in the end stages, fibrosis may become extreme. Perhaps the most interesting changes are the cystic ones, which may vary from a few fine scattered cystic reticulations through generalized emphysema to bullous degeneration or advanced honeycombing. Cysts containing fluid which alternately fill and empty, thus presenting a striking picture on serial x-rays, have been reported.[35] The effects of gross bronchial obstruction, with localized emphysema followed by atelectasis and pneumonitis, may be noted. There may be extensive mediastinal and hilar lymphadenopathy.[25]

No characteristic laboratory findings have been noted. Blood counts occasionally reveal a transient eosinophilia. Bronchoscopy in our case and in one of Mazziotti's revealed diffuse mucosal erythema.

Eosinophilic granuloma of the lung must be differentiated from tuberculosis; sarcoidosis; silicosis, asbestosis, and other pneumoconioses; coccidioidomycosis, histoplasmosis, and blastomycosis; Hodgkin's disease and other lymphomas; and the various types of fibrocystic disease of the lung including congenital fibrocystic disease associated with pancreatic fibrosis, and idiopathic pulmonary fibrosis with emphysema. When history, sputum studies, skin tests, serologic tests, roentgenograms, bronchoscopy, and node or fat pad biopsies have failed to yield a definitive answer, then lung biopsy, a relatively innocuous procedure, should be performed in order to make a conclusive histologic diagnosis. We have preferred a small formal anterolateral thoracotomy under intravenous Pentothal anesthesia with endotracheal intubation and oxygen administration, for more adequate examination of the lung and more accurate selection of tissue to be biopsied. However, the simplicity of Klassen's[55] method of lung biopsy has much to recommend it.

There is no established effective therapy for eosinophilic granuloma of the lung The classical modality is x ray therapy, and although good results have been reported with its use,[4, 51, 103] the effect has usually not been striking[16, 23, 58] ACTH and cortisone should theoretically reduce the extent of the interstitial fibrosis,[6, 9] but little actual improvement has been noted following their use[8, 11, 48, 58, 68] Chemotherapy with nitrogen mustards has been advocated[9] Perhaps the most recent approach to this problem is that of antibiotic therapy Aronson[5] reports remarkable clearing of the process of Letterer Siwe's disease with streptomycin therapy, and Brerman and associates report amelioration in the same disease using combined antibiotics In the case reported herein, slight clearing followed antibiotic administration It must of course be recognized that natural remissions have been reported without therapy[58, 79, 104] Since the disease is generalized, there can never be any indication for primary pulmonary resection However, palliative surgery may occasionally be considered in selected cases of bronchial stenosis or obturation of the bronchus with granulomatous tissue, in cases of giant bullous degeneration of the lungs, and in cases of repeated spontaneous pneumothorax with incomplete re expansion of the lung[8, 66a]

The prognosis must be guarded in all cases In general, the older the patient at the time of onset, the more benign is the course While the prognosis is apparently better when only one tissue is involved, observation over a prolonged period of time is necessary to rule out the onset of foci elsewhere A progressively severe course may usually be anticipated, with increasing pulmonary fibrosis and cystic transformation of the lung, and terminal cor pulmonale Nevertheless, the disease may remain stationary for long periods of time, and spontaneous remission may occur Cases of generalized systemic involvement with pulmonary lesions remaining asymptomatic for as long as ten years after onset have been reported[98] The prognosis for primary pulmonary lesions cannot be stated

CONCLUSIONS

1 A case of primary eosinophilic granuloma of the lung without other demonstrable systemic involvement is presented, representing, we believe, the fifth reported case

2 The disease is characterized by striking fibrotic and cystic pulmonary degeneration

3 Eosinophilic granuloma should be considered in the differential diagnosis of nodular, infiltrative, and fibrocystic disease of the lung

4 Lung biopsy is an essential diagnostic procedure in such cases

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INDUCTION OF BRONCHIAL CANCER BY LOCAL MASSING OF CARCINOGEN CONCENTRATE IN OUTDRIFTING MUCUS

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INHALED CARCINOGENS

THE danger in inhaling cigarette smoke lies in the smallness of the particles, for this ensures that many if not most of them are carried to the rich capillary net of the delicate alveolar walls where the diffusible product is immediately absorbed to give the smoker his "kick." It is to get this effect that he inhales. If the smoke is held in the lungs long enough there is little or none of it visible in the exhalation. But not all of the elements of the smoke are thus promptly absorbed, and tarry ultramicroscopic materials remain on the alveolar walls, doubtless temporarily entrapped in the alveolar mucoid fluid (Macklin[8, 9]), and are speedily accumulated in the segregation apparatus of the phagocytic pneumonocytes (alveolar phagocytes). These "dust cells" make their way to the glottis to be swallowed or spat out. They are of the same category as heart failure cells. In smoker's sputum their segregation granules appear brownish in color. The dust cells probably never burrow out of the alveoli to enter the lung interstices and lymphatic vessels as some writers used to suppose (Macklin[10]). But only a fraction of the dust cells reach the glottis intact; most of them disintegrate and their contents are borne along in the magic carpet or raft of mucus that constantly moves from the alveolar region along the bronchial system to the trachea and glottis. This carpet is motivated by millions of underlying ciliary fingers waving sedulously outward. It is a vital escalator for without it the lungs would soon ill with inspired dust.

Now the smoke derivatives, thus infesting the surface of the lung interior, both outside and inside the dust cells, are carcinogenic. Wynder, Graham, and Croninger[4] have told us how cigarette smoke residue, painted on live mouse skin, induces cancer there. The mucus carpet is charged with this carcinogen. Part of it doubtless never enters the dust cells at all but is picked out of the smoke by the sticky bronchial walls before it enters the alveoli. Graham[4a] has pointed out that 90 per cent of all bronchial cancers are of the epidermoid (squamous-cell) type and that it is very rare in nonsmokers.

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THE TRIANGULAR ESCALATOR

To visualize the diagrammatically deltoid form of this gliding mucus raft which may be so dangerously infiltrated with cancer-producing eliminates, we must imagine the bronchial tree as split open, its walls flattened out, and sections of equivalent caliber laid side by side (Macklin and Macklin[12]). This feat would be impossible practically, but in imagination it may be accomplished. It is dramatically clear that the peripheral alveolar part of this field is immensely greater than the hilar part, being one hundred square meters or one million square centimeters in area (von Hayek[14]) as compared with but a few square centimeters. With such a discrepancy, we would be unable to represent the two in a realistic drawing, but by imaginatively shrinking the peripheral part we may construct a practical diagram, as has been done in Fig. 1, which epitomizes the main point of this discussion, namely, that as the carpet moves from base to apex of the triangle its streams converge and its contents become concentrated. By using ellipsoidal contours, an attempt has been made in Fig. 1 to suggest the enormously greater area of the peripheral region as contrasted with the stem, so that instead of a triangle one has something more like the transection of a renal pyramid.

CONVERGING CURRENTS

The peripheral alveolar zone is, in inhalers, the main "catchment basin" of smoke-carried carcinogens. It is composed of all the alveolar air surfaces put together. In cigarette smoke inhalation, a heavy shower of tarry particles falls upon it. These are really smog particles for, from the warm humid air of the respiratory tract, each grain gathers up an investment of water molecules. These grains, after impingement on the alveolar wall, are moved centrally, and it would seem mainly within dust cells and probably by amoeboid movement, first to the stems of the lung lobules constituted of the transitional (respiratory) bronchioles (Macklin[9]). This first lap of the journey has entailed the most spectacular narrowing of the carpet which, up to this point, has been aqueous-mucoid (Macklin[9]) rather than mucous in type. There is no apparent ciliary motivation here.

THE VULNERABLE HILAR EPITHELIUM

We may, then, imagine this gradually narrowing raft, in which lurk carcinogens, as drifting from the periphery of the lung toward and into the hilus. Each part of it must pass consecutively along the alveolar bronchioles (ducts, Macklin[9]), transitional (respiratory) bronchioles, various orders of smooth-walled bronchioles, and then the small, medium, and large bronchi. The reason the raft is continuously narrowing is because it has to adapt itself to the extent of the wall space of each class of air tube and this collective wall area is reduced rapidly. The collective cross section and hence width of the spread-out wall mucosa of all the small bronchi, for instance, is greater than that of all the medium bronchi and, in like mode, there is surface diminution on down to the trunk of the bronchial tree. We may ask why the raft does not

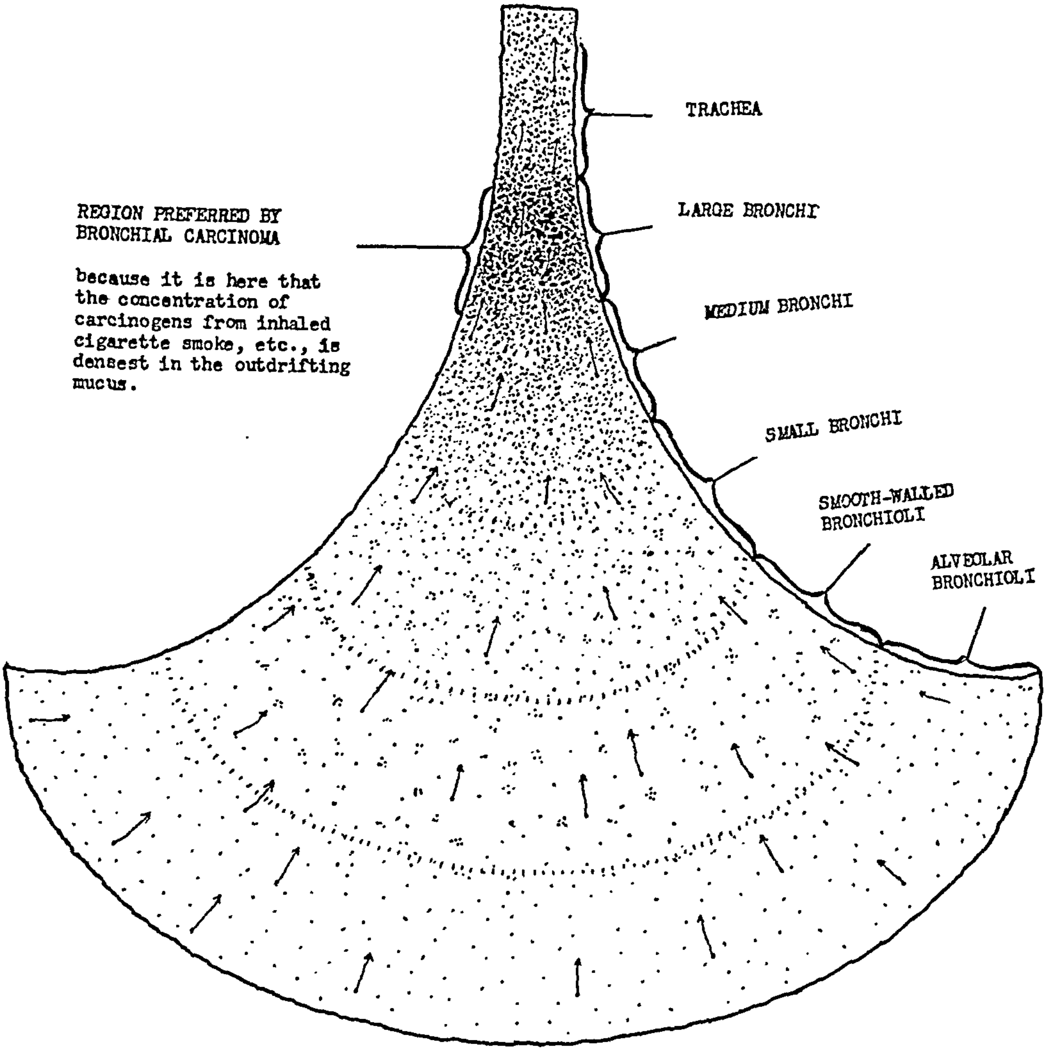
TRACHEA
LARGE BRONCHI
MEDIUM BRONCHI
SMALL BRONCHI
SMOOTH-WALLED BRONCHIOLI
ALVEOLAR BRONCHIOLI
REGION PREFERRED BY BRONCHIAL CARCINOMA
because it is here that the concentration of carcinogens from inhaled cigarette smoke, etc., is densest in the outdrifting mucus.

Fig. 1.—The "Carcinogenshed." An idealized conception of the surface drainage field of the interior of the lungs to illustrate the convergence of mucus currents with consequent concentration of the contained carcinogens of inhaled cigarette smoke, smog, radioactive "fallout," radioactive mine dust, etc. In the large bronchi, there is great concentration combined with slowing of the mucus raft, and here the epithelium is exposed to a maximum of carcinogenic action and bronchial carcinoma is most abundant. Concentration of carcinogens may be increased by dehydration as the raft of mucus moves over the conjoined bronchial area, in the direction indicated by the arrows, toward the hilus of the lung and ultimately the glottis. Although zones are marked off for the alveolated bronchioli (representing the alveoli), for the smooth-walled bronchioli, and the small, medium, and large bronchi, there is no attempt to represent these areas on a proportional basis. For instance, the outer alveolar zone would have to be immensely greater than it is to bring it into true relation to the area of the large bronchi. Most of the carcinogen-bearing smoke and smog falls on this great peripheral alveolar territory; its walls are not covered by true mucus but rather by alveolar mucoid fluid (Macklin[8, 9]), and no true cilia abound here so that transport of carcinogens seems to be largely intracellular and amoeboid in type. The dots indicate the carcinogens moving in the direction of the arrows. The broken transverse crescentic lines stand for the carinae of the large bronchi and trachea. This diagram is, of course, not intended as a universal indicator of the sites of bronchial cancer for not a few of these occur peripherally to the hilar region of the lung. As a watershed gathers in and drains away water so this "particleshed" gathers in and drains away carcinogens that have fallen upon it from the inspired air.

greatly thicken as it narrows, even to the extent of obstructing or blocking the tubes, particularly as it must gain volume from added secretions, and the explanation seems to be that it gives off water as it moves along (for alveolar air may lose water to alveolar fluid and be desaturated, Macklin[11]). Such a "boiling down" would further condense the carcinogens. Therefore, in the larger bronchi of the hilar region the carcinogens are tremendously concentrated. That is why the epithelial cells of the large bronchi, underneath this raft, are exposed more than are those of any other part of the bronchial tree to the delinquency-promoting action of carcinogens on cells. That is why most bronchial carcinoma develops in the hilar region. By the time the raft gets to the hilus, its diminution in area by convergence is as much as ten thousand into one.

SLOWING AT CONFLUENCES

Another factor making for heightened carcinogenic action in the hilar region arises from the fact that at the great bronchial confluences there is an inevitable lateral shift of much of the mucus current entailing a thickening of the mass at such points and delay in its onward progress. This makes these forkings the scene of extra accumulation and also a lingering of the carcinogen forces which then have leisure to act with great strength upon the underlying epithelium. This will be clear when we recall that the two streams of the inner walls of the bronchi which merge at a fork come to an end, so to speak, at the edge of their carina and can only proceed by turning out along the curving keel. Any illustration of the human trachea and large bronchi (von Hayek[15]) will show that the hilus of each lung is a confluence region with as many as four carinae. It is not difficult to see why the mucus currents here should slow down, for the ciliary burden must become much heavier rather suddenly from convergence of currents and deviations at carinae. Another point to be mentioned about the hilar region in this connection involves specially the bronchi to the upper lobes at the points where they enter the main bronchi and that is, that the mucus traveling down the medial side has to make a sudden turn as it climbs up the lateral wall of the main bronchus, and because of the upright posture it is moving against gravity. It is like walking on the ceiling. This site is said to be a favorite one for bronchial cancer, particularly on the right side. My readers are enjoined to consult a bronchial map (Boyden[2]) and try to figure out the course of masses of mucus proceeding along any of the branches centering in the hilus. When this visualization is done in three dimensions and attention is focused on the inside of the tubes, it will be realized that there is here (1) a rather sudden merging of currents of mucus entailing a thickening of the raft with an inevitable increase of the strain on the underlying cilia, which cannot possibly be reinforced in any way to meet it, and (2) a complex of devious courses of mucus streams as they curve out to avoid "falling over the edges" of the carinae and, thus, they undergo unavoidable accumulation and retardation in the process. If now they will see in this mucus coating of the large bronchi a hotbed of carcinogens, they will be educated to an understanding of the basic reason for the hilus being par excellence the part of the entire field of the bronchial epithelium where carcinomatous change occurs. Why masses of mucus arriving at a carina come to the end of the road, so to speak, and are unable to go straight on, but must be deflected by special ciliary arrangement to one or other side is even more convincingly demonstrated by looking into the interior of a living bronchus through a bronchoscope; but a good substitute for this experience is had by viewing illustrations of bronchoscopic fields such as those of Fig. 125 of Brock.[3]

Then, too, it is more than possible that this long-acting concentrate of smoke products would functionally depress the oversmeared ciliary propellant making the current sluggish and the carcinomatous raft to lag, particularly in the hilar region. Ciliary paralysis, partial or complete, would thus constitute an accessory retarding influence. Metaplastic change even removes the cilia. All told, it seems very probable that in the hilar region of the bronchial tree there is more cancer-producing substance acting on the epithelium for a longer time than there is anywhere else in the air tract. The effective carcinogenous material is probably not intracellular; it is more likely that it is in the remains of phagocytes which carried it out of the alveoli and along the alveolar bronchioles (ducts) to the ciliated part of the air tract. Indeed, some of the material may never have been phagocytized at all but have been intercepted as particles by the sticky mucus of the raft. The carcinogen acts doubtless as a molecular solution upon the epithelium. Carcinogenic power probably varies directly with the concentration.

SMOGS AND RADIOACTIVE DUSTS

Inhaled cigarette smoke has been widely incriminated in bronchial cancer induction, but it is surely not the only offender, and the special vulnerability of the epithelium of the hilar bronchi to cancer must be related to all dusts and smogs containing carcinogens borne in the mucus currents passing over it. The mechanism would be the same for all. The outdrifting mucus raft affords the *only means* of eliminating particulate matter admitted to the lungs save that available for large masses when peristaltoid movements of the bronchial musculature (Macklin[7]) and coughing come into play. The air of great cities like Los Angeles is contaminated with the products of combustion of coal and oil and these, especially those of oil, are in very finely particulate form and attract water molecules to form smog. These minute smog particles are likely to travel right to the alveoli and impinge on the walls thereof. It is probable that smog always forms when very small particles are being carried along in the humid air of the bronchial tree. The danger of cancer induction is enormously increased if the particles are radioactive, as was the case in the dusty mines of Schneeberg and Joachimsthal (Lorenz[6]) where so many of the miners became victims of bronchial carcinoma. This breathing in of "hot" particles in certain unprotected industries is an invitation to lung cancer. Entire populations, in these days of the testing of thermonuclear weapons, may be subjected to this hazard, for the inhalation of relatively small quantities of radioactive "fall-out" may ultimately result in a great crop of bronchial cancers. This could occur, of course, only in those people whose intake of "hot" dust was prolonged and insufficient at any one time to induce immediate serious illness. The conveyance of these radioactive particles in the mucus of the air tract and the concentration of the carcinogen in the hilar region would be like that described for inhaled cigarette smoke. It may take some thirty years or more of action of carcinogen concentrate from inhaled cigarette smoke to break down the contracancer defences in bronchial epithelial cells (Graham[4a]), but that from frequent exposure to radioactive fall-out would likely require less time. Chronicity is of the essence of carcinoma induction. Primary origin is doubtless in fields of epithelium, with progressive conversion of normal into cancerous cells, rather than in single cells or small foci of cells (Willis[16]), and there may be more than one such field (Ackerman[1]). The exact reason for the selection of any special field for cancerous change is not apparent. It may be a local hereditary predisposition. The same explanation may apply to those bronchial carcinomas which occur beyond the hilus; but here the exciting cause would be found, as for the others, in the chronic application of carcinogen concentrate. Slaughter and his associates have recently discussed field cancerization and multicentric origin.[13]

TRACHEA

Why is the trachea less involved in cancer than are the large bronchi? Certainly it bears the end result of convergence of mucus currents and desiccation of the carcinogen-infiltrated mucus. Possibly it is because the mucus raft travels more rapidly in the straight trachea, and is not abruptly piled up and slowed as in the devious course of the hilar bronchi; and is not on the whole so adversely affected by gravitational influence as are certain parts of the large bronchi; and possibly because its cilia are not overburdened and are more efficient. Certainly there is not at the origin of the trachea as sudden and great an augmentation of the ciliary load as there is in the hilar region, as the cross section of the two main bronchial tributaries is not much different from that of the trachea. If one of the alternative explanations for the prevalence of hilar bronchial cancer be applied here, namely, that it is due to some sort of "heat stroke" by the entering column of hot smoke, the trachea should succumb before the bronchi and even this guess would apply only to smokers. If the cancer-producing action were primarily on the first part of the trachea more heavily involved than the great bronchi. By the time the mucus gets to the trachea it is doubtless depleted in carcinogens.

PREVENTION

For the cigarette smoker, the ideal course would be total abstinence from cigarettes; but short of this it is wise to exercise moderation and particularly to abstain from inhaling the smoke. With the dangerous smogs of cities, radioactive mine dusts, and "fall-out," elimination of the cause is the essential thing. Masks of proper type will aid in emergencies. Education of the public (Hudson[5]) will cut down the rate of which is already the most frequent carcinoma in the human male (Graham[4a]). Storm warnings are flying. Mayer[13] has affirmed that, at present acceleration, the death rate from lung cancer is expected in the next quarter of a century to exceed that from *all other cancers combined.*

SUMMARY

The smoker who inhales brings by far the most extensive area of his lungs, the alveolar, into play to catch carcinogens. These smoke particles, deposited on the inner surface of alveoli and air tract, are returned to the pharynx in a layer of moving mucus which converges and lingers in the hilar region. Here the carcinogens are specially concentrated and stagnated, and they act with high intensity and chronicity on the epithelium of the great bronchi, to induce more cancer than elsewhere in the bronchial tree. Other carcinogenic particles, in smogs and radioactive dusts, have a similar mode of application to the bronchial epithelium with disastrous result.

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PHYSIOLOGIC ASPECTS OF BRONCHOGENIC CARCINOMA

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AT the present time, the treatment of choice for bronchogenic carcinoma is extensive surgery, the only approach which offers the possibility of cure Primarily, as a consequence of the problems posed by bronchogenic carcinoma, the technical aspects of thoracic surgery have been intensively studied during the past two decades Surgical techniques, as well as operative and postoperative procedures, have been developed to the point that pneumonectomies are common operations in all our large hospitals Many of the successful operations in the past, however, have resulted in complete respiratory crippling of the patient Thus, the physiologic problem of over all pulmonary function and pulmonary reserve has become of critical importance In fact, lack of adequate pulmonary reserve is probably the only major contraindication to pneumonectomy at present, outside of the extension and metastasis of the tumor itself

This situation has been a great stimulus to the study of pulmonary function As a result, methods have been developed to determine pulmonary reserve, and even to estimate with some accuracy what the respiratory status of a patient will be after surgery These procedures are not limited to use in large university research laboratories, many of them are sufficiently simple and inexpensive to be utilized in any hospital that has facilities for surgical exploration of the chest Such testing, of course, is time consuming and some training of the examiner is necessary It often provides clinically valid answers to the questions important to surgical evaluation, and the value in prognosis warrants the time and effort required

COMMON PULMONARY FUNCTION TESTS

The requirements of physiologic testing vary considerably from patient to patient Sometimes a few simple studies will provide clear and unequivocal information, while a more thorough evaluation is needed for some patients The purpose of the following description is to define the basic minimum study necessary to prognosis in the easier cases, such a study will also serve as groundwork for more detailed evaluation in the difficult cases

Basic Ventilatory Tests—Simple ventilatory tests should be done at the time of diagnosis[1] Maximum breathing capacity has been so well standardized

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that it is a useful point of reference, even though in certain respects it may be considered somewhat unphysiologic. Some form of resting, exercise and recovery ventilatory test provides valuable information. These tests can be correlated to give various ventilatory coefficients. Spirographic tracings are informative, since they show over-all compliance and volume changes, both qualitative and quantitative. These tests require simple instruments and only a minimum of training on the part of the operator. Tests, such as breath holding, pneumatometry, and others, are used by some workers to amplify results further.

Radiologic Techniques.—The usefulness of fluoroscopic study of the heart silhouette is generally recognized. Such studies are usually amplified by electrocardiograms and determinations of hematocrit, venous pressure and circulation time. These evaluations are particularly important when a pneumonectomy is contemplated, since right-sided heart failure may ensue in patients with emphysema in the remaining lung and any significant degree of pulmonary hypertension.

Fluoroscopy is also useful in detecting specific abnormalities affecting pulmonary function, such as a weak diaphragm. Inspiration-expiration posteroanterior x-rays of the chest should be correlated with vital capacity tests. While measurement of lung areas, as shown in x-rays, with a planimeter does not take into account the effect of pathologic lung changes, it does give a rough basis for differentiating the ventilation volume of each lung and for estimating residual volume.[2]

Radiologic information can be supplemented by simultaneous intrapleural pressure readings, using standard pneumothorax equipment, during quiet and maximal respiration. These data are particularly useful in determining pulmonary compliance on both sides.

Intrapulmonary Gas Mixing Tests.—The study of residual air and intrapulmonary gas mixing has become a common procedure in many hospitals. Either the closed-circuit helium or the open-circuit nitrogen method will provide information as to the presence of overdistention and the mixing of inspired air with alveolar gas. While these tests permit a more accurate definition of these aspects of pathophysiology than do radiologic methods, they do not differentiate between the two lungs, and hence are, we feel, more applicable to the analysis of a diffuse disease process than to the localized process of a cancer. They may, however, contribute to prognosis after differentiation has been established by other techniques.

Bronchospirometry.—Although bronchospirometry is the best single differentiating function test now available, the discomfort it causes the patient and the long testing period have limited its use. Also some training is required for correct evaluation of bronchospirometric findings with the problems of catheter blockage and the effects of the catheter itself upon the air flow. Since it is the most accurate technique we have for determining volume ventilation, oxygen consumption, and intrapulmonary mixing for each lung separately, the value of bronchospirometry outweighs these difficulties in practically every instance in which the results of simpler tests are ambiguous. It is especially valuable in assessing the condition of a single lobe in cases where a choice must be made between a lobectomy or a pneumonectomy involving the sacrifice of normal tissue. Studies should always be made during exercise (with foot pedals) as well as at rest.[3]

It must be remembered that bronchospirometry can evaluate the function of a lung or a lobe only under the conditions prevailing at the time of the examination. Predictions of postoperative course are often found to be conservative because they do not take into account the great adaptiveness of the organism to the postoperative situation.

Blood Studies.—Many of the simpler studies involving arterial oxygen saturation can be done with a photoelectric ear oximeter. The clinical estimate of oxygen in the blood, however, requires experience. Isolated arterial oxygen values, of course, are of little significance, but desaturation on exercise is important to know, particularly when it can be correlated with oxygen consumption.[4] The breathing of pure oxygen may demonstrate abnormalities of gas distribution and blood perfusion.[5]

The detection of respiratory acidosis, as reflected by arterial CO_2 tension, is important during all stages of operative treatment—preoperatively, during the operation, and particularly in the immediate postoperative period. In most laboratories this value can be readily ascertained by determining the arterial blood pH and the direct plasma CO_2 content, calculating the tension by the Henderson-Hesselbalch equation. The Van Slyke apparatus is found everywhere, pH meters are coming into widespread use, and the range of their serviceability fully justifies their purchase. Arterial puncture is a simple procedure that should involve no hazard.

Simultaneous blood-gas studies, with calculation of venous admixture, diffusion gradients, and so forth,[6] are beyond the scope of most hospital laboratories. Such studies are not always as informative in determining pulmonary function in cases of bronchogenic carcinoma as in cases of diffuse disease.

Admittedly, lung function tests are not as well standardized as routine clinical use makes desirable, and often the clinician must work with improvised equipment. However, the information gained, even with the most limited materials, can aid in determining the type of surgical procedure to be undertaken and even, in some cases, whether an operation is feasible. They also are a useful guide to prognosis of postoperative lung function, and can alert the surgeon to operative and postoperative hazards.

THE EVALUATION OF PRE-EXISTING PULMONARY ABNORMALITIES

Pre-existing pulmonary abnormality is very common in patients in the age group in which bronchogenic carcinoma is most frequent. Some degree of emphysema is almost the rule; in addition, such disorders as fibrosis and pleural thickening are often encountered. These abnormalities introduce special diagnostic problems because of the difficulty of isolating the effects of the tumor from the general pulmonary insufficiency.

In some cases, a pre-existing abnormality is easily recognized. Obstructive emphysema (not due to the tumor) is manifested by the typical delay in expiration as shown on the spirogram, as well as by the increase of residual air in relation to vital capacity. Trapping of the "normal" lung by pleural fibrosis is also easy to detect but, in many instances, it is difficult to decide whether there is any pulmonary insufficiency in addition to that directly due to the tumor. It is dangerous to assume that the nonaffected side is normal, especially when radiologic or clinical evidence suggests abnormality. If spirographic tracings do not give volume and velocity measurements which are at least one half of the predicted value, some degree of ventilatory difficulty on the normal side should be presumed. Also, abnormal intrapleural pressures on the contralateral side confirm the presence of a generalized pathologic condition in addition to the tumor.

Even when abnormality of the other lung has been established, the degree of respiratory embarrassment due to that lung may be difficult to determine. Occasionally this problem can be approached by trying to approximate the degree of embarrassment due to the tumor and its secondary manifestations, and subtracting this from the total respiratory deficiency. More often, conditions secondary to the tumor will be impossible to estimate; even where there is clear evidence of emphysema, it may be difficult to tell whether it is compensatory or independent of the tumor, or whether it is reversible or not. A study of the area change in posteroanterior x-ray films made during inspiration and expiration may give a reasonable basis for determining the volume ventilation of the contralateral lung; however, in many cases, bronchospirometry will be very nearly mandatory. Every effort should be made to determine the efficiency of the other lung, even if it involves referring the patient to a better-equipped institution; otherwise there is the risk that successful surgery may actually shorten the patient's life.

A good many investigators have attempted to develop more accurate methods of evaluation. For example, angiocardiography has been used to demonstrate not only blood flow through atelectatic tissue, but also through anastomotic channels in the tumor mass.[7] Cardiac catheterization, using a balloon-tipped catheter, has been applied in determining the effects of blocking pulmonary artery blood flow to an affected lobe. There have been attempts to forecast partially the effects of possible pneumonectomy by blocking simultaneously both blood and air flow to the lung to be removed.[8] Although these procedures may hold out hope for the future, they are too complicated and too hazardous for clinical application at the present time.

It should be pointed out that in a good many instances the elementary methods of determining respiratory function efficiency will suffice to clear up doubts about the feasibility of an operation. This is particularly true in those cases in which severe exertional dyspnea seems out of proportion to the amount of damage indicated by lung function testing or visible radiologically. In many cases of lung cancer, the subjective feelings of dyspnea bear almost no relation to the real extent of abnormality. This is probably a result of increased stretch reflex irritability in an area of "stiffened lung" due to infiltration. In such cases, other lung function tests will show virtually normal values.

PULMONARY FUNCTION TESTS DURING THERAPY

Surgical procedures which involve only the resection of damaged tissue are likely to result in improved lung function by improving the distribution of blood and gas in the lungs as a whole. In such cases pulmonary function is rarely a problem after the operative and postoperative phases. However, in some instances where preoperative studies have revealed only a well-localized peripheral tumor, evidence of spread at thoracotomy may call for pneumonectomy. This will sacrifice a good deal of lung tissue, reduce ventilation, and shunt the whole of the cardiac output through the remaining lung. If there is already gross obstructive emphysema with respiratory acidosis, the patient may be unable to ventilate sufficiently after the operation. If there is pulmonary hypertension with early signs of right ventricular strain, the pneumonectomy may be enough to precipitate right heart failure.

If pulmonary reserve is adequate and an operation is undertaken, the patient's respiration must be watched carefully during the operation and particularly postoperatively. Ventilation during the operation is usually not a problem, since it is controlled by the anesthetists with a direct view of the thorax, although even in this situation respiratory acidosis may occur unless special attention is paid to tidal volume. In some of the better-equipped hospitals the end-expiratory CO_2 content is continuously followed by automatic analyzers, so that the smallest degree of acidosis can be detected and checked.[9] In institutions without this equipment, a periodic check of arterial CO_2 tension can be made by analysis of CO_2 content and pH values.

After the thorax is closed, but before the patient completely recovers consciousness, respiration is liable to become too shallow to maintain adequate tidal volume. In the past there has been a tendency to administer oxygen to such patients, which treats the hypoxia selectively and masks impending respiratory acidosis. It is our conviction that the intermittent positive pressure apparatus is preferable in respiratory failures of this kind, since it restores adequate ventilation, thereby preventing the accumulation of CO_2 as well as ensuring normal oxygen content in the alveoli.

A number of other postoperative developments such as pleural effusions, atelectasis, paradoxical respiration, and pericardial tamponade may tend to diminish respiratory efficiency. In our experience, painful respiration, usually at the site of incision, has been the most frequent cause of respiratory embarrassment. It can usually be controlled by a paravertebral alcohol block or Novocain infiltration of the incision.

In the later postoperative phases, scoliosis is perhaps the greatest danger, especially after pneumonectomy with thorocoplasty. The occurrence of scoliosis, which may mean the difference between respiratory crippling and a reasonably normal life, can be prevented only by diligent physical therapy. It has been our practice to institute such therapy before surgery and continue it for about six weeks, until the patient is able to carry on the exercises by himself.

Last, it should be pointed out that palliative radiotherapy should not be contemplated in patients with any degree of respiratory embarrassment unless pulmonary function tests are performed. It must be remembered that such therapy results in immediate local swelling and edema which may be followed by fibrosis involving a good deal of normal tissue. The end product is devascularization, focal emphysema, and loss of alveoli, with a consequent reduction in the pulmonary capillary bed and a diminished alveolar surface area. If there is any considerable pulmonary insufficiency to begin with, radiotherapy can easily result in making a patient a respiratory cripple.

SUMMARY

The surgical therapy of bronchogenic carcinoma demands preoperative study of pulmonary function. In many cases simple ventilation studies and inspiratory-expiratory posteroanterior x-rays are sufficient. In difficult cases where further study is required, differential bronchospirometry is the most valuable procedure. The isolation of the effects of the tumor from pre-existing and consequent abnormalities may be very difficult.

Inadequate ventilation during surgery and in the early postoperative phase may readily occur. Pain in the chest may restrict ventilation. These factors should be watched for. Scoliosis may develop later and be of sufficient degree to restrict ventilation. Radiotherapy may markedly interfere with pulmonary function.

Detection, prevention, and energetic therapy of causes of pulmonary insufficiency are mandatory for best results in the surgical treatment of bronchogenic carcinoma.

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Original Communications

THE USE OF SHUNTS IN SURGERY OF THE THORACIC AORTA

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IN CONGENITAL or acquired constrictive disease of the arterial system, an enlarged collateral circulation is present, but this is not true in the case of aneurysms The circulatory shunt is an immediate surgical substitute for collateral circulation It provides nutrition to distal vital organs and time for meticulous reconstructive surgery The closer the vascular lesion is to the heart the greater the need for a shunt, since momentary interruption of the cerebral circulation disposes the patient to an unnecessary risk In fact, high occlusion of the aorta just distal to the left subclavian artery is not without risk since marked physiologic disturbances are precipitated not only upon occlusion but upon release of the occlusion as well

Sudden occlusion near the arch produces a proximal hypertension which, if sustained, may prove to be a severe imposition upon both the left ventricle of the heart and the cerebral blood vessels It is reasonable to believe that in older patients the sudden additional vascular tension may precipitate a cerebral accident or, if prolonged, dispose the patient to myocardial insufficiency because the status of the coronary arteries is usually an unknown factor However, cardiac failure is unlikely to occur if the coronary blood supply is capable of increasing in proportion to the myocardial demands

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Sudden release of the occlusion is more hazardous than sudden occlusion because coronary flow is reduced at a moment when myocardial demand is maximum. To wit, it is known that distal to the occlusion a hypotension develops with an associated hypoxia and vasodilatation. Upon sudden release of the occlusion, "hemorrhage" may be said to occur into the dilated vascular bed. A marked fall in blood pressure often results which is tolerated well by all the various systems except the vital coronary system whose flow is momentarily decreased in the presence of a compensatory increase in the work of the heart. Stagnation of the blood under these circumstances causes reduction in the volume of circulating blood with a decrease in blood returning to the heart. The reduced cardiac return is accountable for the low cardiac output and the fall in both systolic and diastolic pressures. Since the diastolic pressure is an important adjunct to coronary flow the stage has been subtly set for a cardiac catastrophe. After high occlusion of the aorta for perhaps an hour, the heart, laboring against a head pressure of this degree, is approaching exhaustion at a time when the occlusion is suddenly released. The strained heart in a desperate effort to compensate for the sudden change in vascular dynamics is then called upon to make a final heroic effort to restore circulatory physiology to normal.

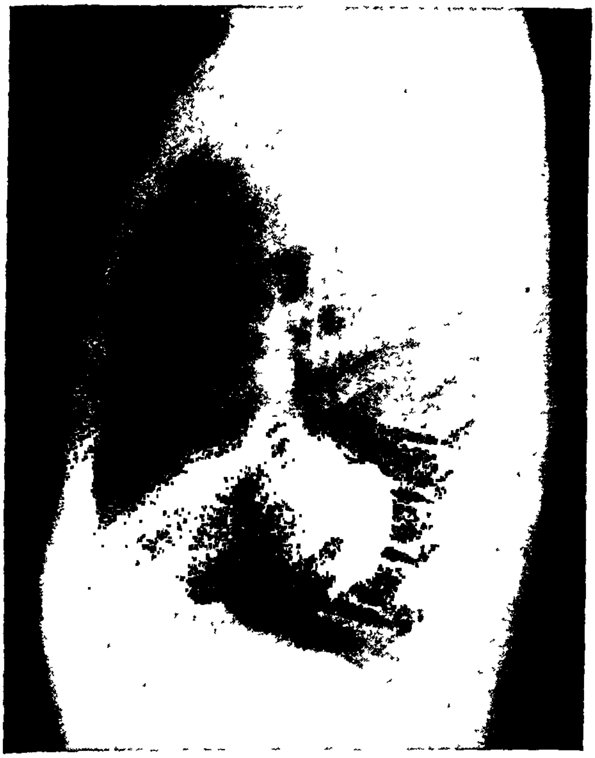

Fig. 1.—Left lateral film taken in 1949 revealed a mediastinal mass, posterior to the heart, proved on exploration to be an aneurysm of the descending aorta. A reproduction of posteroanterior film of chest is omitted because it was uninformative.

It is the knowledge of this potential disaster that drives the surgeon to a hasty conclusion of a rather meticulous piece of surgery. He knows that in his contest with time he must frugally protect the cardiac reserve by reducing to a minimum the hypertensive and hypotensive intervals Should prolonged occlusion prove necessary, the integrity of the coronary circulation may be enhanced by the gradual release of the occlusion and the simultaneous use of a vasoconstricting drug These hazards are avoided by the use of a shunt

The following is a case report in which multiple aneurysms of the thoracic aorta were resected while circulation was maintained with a heterologous aortic external shunt

CASE REPORT

L C, No 45055, a 63 year old white male cook, was admitted to the Brooklyn Veterans Administration Hospital on Aug 23, 1954, for evaluation of the progressive increase in the size of thoracic aneurysms as demonstrated by serial chest x rays

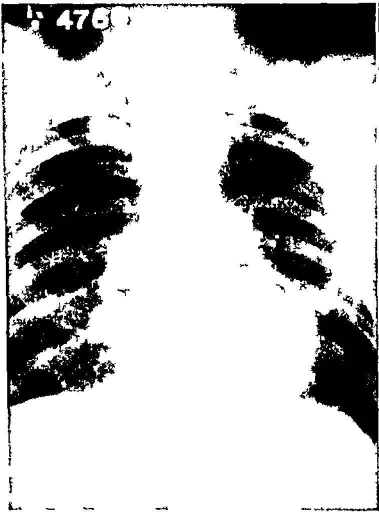

Fig 2

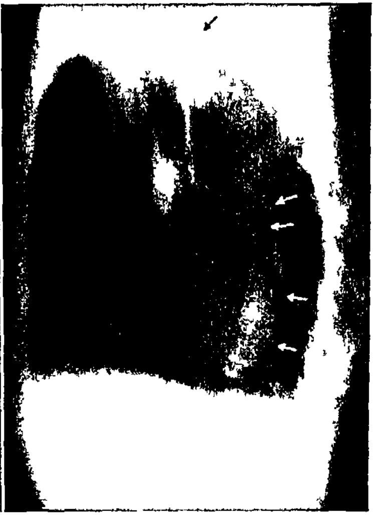

Fig 3

Fig 2 —Posteroanterior chest film taken in November 1954 which revealed 3 aneurysms

Fig 3 —Left lateral tomogram taken in 1954 shows the aortic pathology in more detail as outlined by arrows The fourth aneurysm in the ascending aorta is not visualized

The aneurysms were first diagnosed in 1949, when the patient presented himself to another institution because of a three month history of a productive cough Work up revealed a mediastinal mass (Fig 1) On exploratory thoracotomy on June 1, 1949, two aneurysms measuring 4 cm and 5 5 cm were found in the descending thoracic aorta Oxycel was wrapped about the aneurysms to stimulate fibrosis and the chest closed The patient's condition was fairly satisfactory until February, 1953, when he again was hospitalized for dyspnea, orthopnea, cyanosis, and vertigo Diagnoses of a cardiac decompensation secondary to myocardial infarction and a spontaneous right pneumothorax were made The symptoms subsided on a medical regimen

On admission to this hospital, the patient still had a slight cough and orthopnea. There was no history of syphilitic infection. Physical examination revealed the blood pressure to be 112/70, pulse 70. The chest was emphysematous, with basilar wheezes bilaterally. Peripheral pulses were adequate. Tomography and chest film (Figs. 2 and 3) disclosed a third aneurysm in the arch of the aorta.

An exploratory operation was performed on Nov. 4, 1954, through the bed of the left ninth rib and the fourth interspace (Fig. 4). The first aneurysm (not seen on x-rays), 2 cm., was situated just proximal to the innominate artery; the second, 4 cm., was located just distal to the origin of the subclavian artery; the third, 6 cm., was at the level of the pulmonary hilus; and the fourth, 10 cm., was within 2 cm. of the diaphragm. The aorta was mobilized. Two 25 cm. segments of pig aorta sutured together were made into an external shunt. A cloth-covered Satinsky clamp was placed about the most proximal aneurysm, the dome of the aneurysm excised, and the proximal limb of the shunt sutured into place. The distal limb was sutured into the aorta 2 cm. beyond the distal aneurysm (Fig. 5). In spite of its comfortable length (50 cm.), the shunt did not kink and was easily maneuvered into any necessary position. The descending thoracic aorta, including the three aneurysms distal to the subclavian artery, was resected between clamps and replaced by a 20 cm. reconstituted homograft. The shunt was then removed after having maintained adequate hemodynamics for two hours and fifty-five minutes. The patient tolerated the procedure fairly well, requiring 3,500 c.c. of blood. The vital signs were static except for moderate hypotension with blood pressure ranging between 80 and 130, systolic, and occasional dips to 60 and 70, diastolic.

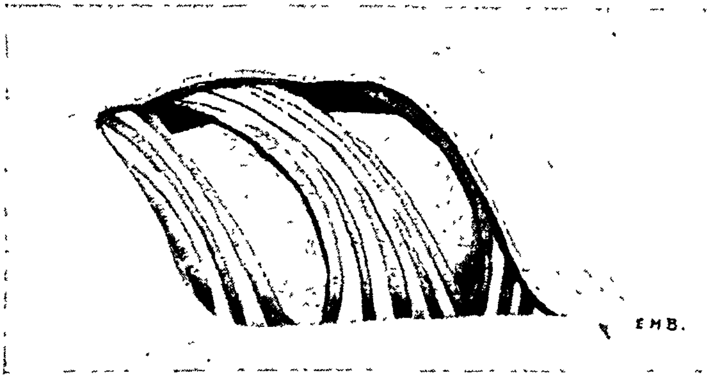

Fig. 4.—Drawing of the approach to the aorta through a long left dorsolateral thoracoabdominal incision. The fourth interspace and resection of the ninth rib are shown.

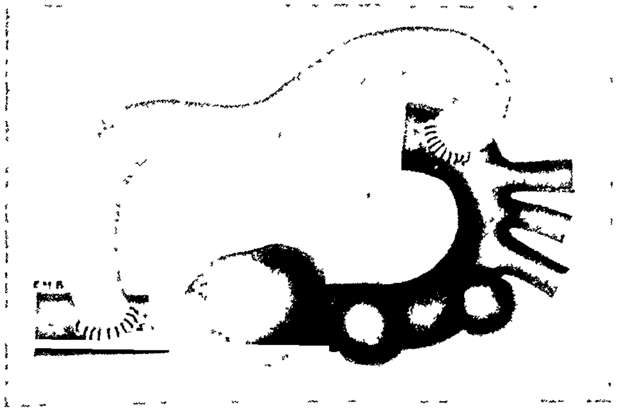

Fig. 5.—Drawing of the aneurysms in the ascending and descending aorta with the heterograft shunt sutured into place distally and into the dome of the first aneurysm proximally.

The postoperative course was marred by memory loss which largely subsided after five days. This was thought to be due to anoxia during the operation. Aphasia, noted for the first few days, and subsequent voice changes were due to paralysis of the left vocal cord. A portion of the wound became infected and cleared after debridement and secondary closure. At the time of discharge from the hospital on Feb. 11, 1955, the patient was fully active. Peripheral pulses were present. Tomography revealed the aorta to be uniform in diameter throughout (Fig. 6). Sections of the specimen revealed the aneurysms to be atherosclerotic.

The patient was readmitted on March 15, 1955, because of anorexia, orthopnea, and exertional dyspnea. The blood pressure was 90/70, pulse 110. There were decreased breath sounds in both bases. Peripheral pulses were unchanged. Chest film (Fig. 7) was essentially negative. Fluoroscopy showed the costal and diaphragmatic movement to be reduced and beneath the diaphragm was a large collection of gas in both the stomach and large bowel. The symptoms were thought to be due to these findings and they subsided on medical regimen. It was interesting to note, on this admission, that there was less pulsation in the grafted segment than in the host aorta.

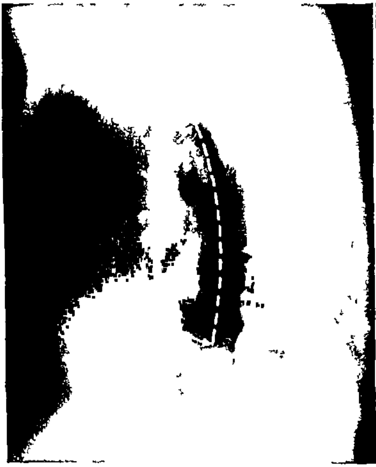

Fig. 6

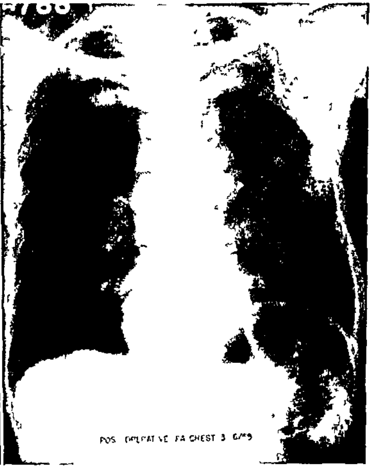

Fig. 7

Fig. 6.—Left lateral tomogram taken three months following resection of multiple aortic aneurysms. The normal aortic contours are 2 mm. posterior to the dotted line.

Fig. 7.—Four-month postoperative posteroanterior film of chest.

HISTORICAL REVIEW

The surgical procedures which have been used for the treatment of aortic aneurysms can be conveniently divided, according to Cooley and DeBakey, into three groups. (1) Those designed to promote thrombosis and organization by ligation, by introduction of foreign material, or by periarterial fibroblastic reaction. (2) Repair of the communication between the aorta proper and the aneurysm while the aorta is temporarily clamped (restorative endo-aneurysmorrhaphy). (3) Extirpation of the lesion with or without restoration of flow through the parent vessel.

1. *Procedures Which Promote Thrombosis.*—Depending on the site of ligation relative to the aneurysm, various procedures were designed for sudden occlusion of the large arteries as described by Antyllus in the second century[78] and then by Anel in 1710, Hunter in the 1760's, and Brasdor in 1798.[79] They failed, among other reasons, because of ischemia distal to the ligation, secondary hemorrhage due to gradual erosion of the suture, and/or recurrence due to loosening of the ligature. The first ligation of the aorta was performed by Sir Astley Cooper in 1817 for an aneurysm of the left iliac artery. From that date until 1920, none of the 18 patients on whom this procedure was done left the hospital alive.[2] Subsequently, survivors were reported but the patients suffered from paralysis or weakness of the lower extremities and/or cardiac decompensation.[2-4] These symptoms subsided when the ligature loosened, which was usually the case.

In order to decrease the morbidity and mortality of sudden occlusion of the aorta, methods of gradual occlusion were suggested so as to stimulate the formation of collaterals and permit the heart to become adjusted to the increased peripheral resistance. These procedures, generally unsatisfactory, varied from occlusion by pressure through the skin to directly applied clamps, ligatures, rubber drains, metal bands, fascia, rubber bands, homologous aortic strips, and intraluminal fascial plugs.[59] Partial aortic occlusion by plication,[87] and incomplete ligature[88, 89] also met with fatal results as, in each group, there was damage to the adjacent tissues, infection, and/or hemorrhage.

This failure gave rise to the idea of wrapping the aneurysms with an agent that would stimulate fibrosis without any immediate partial occlusion. It was presumed that the gradual process of fibroplasia would provide adequate support to the aneurysmal wall. Following Page's[8] lead that polyethylene prepared by DuPont had such properties, Pearse[5] succeeded in producing constriction of the aorta. Cowley and Yeager[9, 10] found the active agent to be dicetyl phosphate. Other investigators[6, 11, 12, 29, 90, 93] along with Cowley and Yeager used this method without uniform success and found that mobilization of the aorta may lead to rupture and that the wrapping may compromise the blood supply of the sac wall. Vargas and Dieterling's[14] method of supporting aneurysms with a nylon net met with similar results.

The third method[27] of producing fibrosis of the aneurysm was introduced by Moore in 1865 who inserted silver wire into thoracic aneurysms to stimulate clotting. In 1879, Carradi passed a galvanic current through similarly placed wires. Blakemore[80] revived this method and found that of 63 patients with syphilitic aneurysms so treated, 34 survived an average of 2.4 years, while 12 died in the hospital. When he[7] added partial occlusion of the proximal aorta with dicetyl phosphate-coated rubber bands, he noted that too rapid occlusion caused claudication in the lower extremities while inadequate constriction resulted in an increased strain (nozzle effect) on the aneurysm with rupture.

2. *Endo-aneurysmorrhaphy.*—The principle of endo-aneurysmorrhaphy with restoration of the lumen of large arteries, as introduced by Matas[1, 81] in 1888, offered the most physiologic approach to the problem up to that time. Bigger[2] reported the first successful application of the method in the treatment

of an aneurysm of the abdominal aorta in a patient in whom an aortic ligature loosened. A similar procedure was used to repair aneurysms of the innominate artery[18] and vena cava–abdominal aorta fistulas.[19, 20] Lane and Pierce[13] successfully combined endo-aneurysymorrhaphy with polyethylene wrapping for an aneurysm of the innominate artery. Woods and Kenney[21] modified this procedure and treated a small aortic aneurysm by infolding the sac and tamponading it against the approximated edges of the defect.

Saccular aneurysms of the aorta lent themselves easily to another modification of this procedure whereby the neck of the sac is clamped and the aortic defect sutured. This has been done unsuccessfully by Tuffier[22] in 1902, and Kummel[23] and Kerstorsky,[76] and successfully by Monod[24] in 1947 and subsequently by others.[6, 25, 26, 91] The importance of excision of the sac to prevent its necrosis and infection was emphasized by Tuffier,[22] Cooley and DeBakey,[6] Bahnson,[25] Lam and Aram,[28] and Freeman and Leeds.[71]

3. *Excision of Aneurysms.*—In dealing with fusiform aneurysms of the aorta, especially above the renal arteries, the previous methods were not always effective or desirable. If this pathology is present, a section of aorta has to be resected and replaced by a graft or prosthesis. Excision of aneurysms of the innominate, subclavian, and carotid arteries with ligation of the parent vessel has been described.[6, 15-17, 25] Alexander and Bryon[38] were the first to report such a procedure on the aorta. In their case, the aorta was ligated just distal to the left subclavian artery and a section of aorta containing a coarctation and an aneurysm was resected. The postoperative course was stormy leaving the patient with residual weakness even though collaterals were developed. Following Crafoord's report[35] that the aorta could be safely cross clamped for 27 minutes while operating for patent ductus (but with damage to the spinal cord after 48 minutes[77]), coarctations of the aorta were resected and the aorta re-anastomosed or grafted.[35-37, 44, 72-74] Neurological sequelae and fatalities were reported in some cases. One cause of several fatalities[30, 31, 42] and possibly another[33] in this latter group, despite the presence of well-developed collaterals, was sudden release of the clamp occluding the aorta. A similar sequel was the result of releasing the occluding clamp, after resecting an abdominal aneurysm[32] and, possibly, after resection of thoracic aneurysms.[34]

In the absence of adequate collaterals, success or failure of excision of aortic aneurysms with homografting depends on the length of time the aorta is occluded. In 1951, Dubost and co-workers[82] reported the successful excision of an abdominal aortic aneurysm and grafting in 1 case. Subsequently, DeBakey and Cooley[39] clamped the abdominal aorta for 48 to 102 minutes while abdominal aneurysms were resected and the aorta grafted, with one fatality after 105 minutes' occlusion. This was followed by their report[34] of the same method for thoracic aneurysms. The aorta was clamped for up to 45 minutes successfully in 1 case, but with residual weakness of the lower extremities in another and with a failure in a case after 77 minutes' occlusion. Hypothermia enabled them to extend occlusion to 62 minutes in a thoracic aneurysm, but there was one death after 53 minutes' occlusion. Under normothermia, Bahnson[25, 40] occluded the aorta just distal to the subclavian artery for 20 minutes without sequelae.

but clamping just proximal to the left carotid artery had to be discontinued after 7 minutes because of hypotension and cardiac irregularity. Further experimental evidence[41-45] revealed that hypothermia lengthened the time that the aorta may be totally occluded but the incidence of complications remained considerable. Investigators have attempted to preserve normal dynamics of circulation in order to avoid the shortcomings of the previously described methods of treatment. This has been accomplished in two ways. In the first, a graft or prosthesis is threaded over a rigid tube that serves as a temporary intraluminal shunt. As early as 1894, Abbe,[46] and Carrel,[47-50] in 1910, pioneered this method when they placed tubes of paraffined rubber, glass, aluminum, and gold plate into major arteries of experimental animals. These tubes functioned as temporary conduits even though they eventually thrombosed. Lately, Hufnagel[51] and others[52-56] successfully used methyl methacrylate, polyethylene, and siliconized stainless steel tubes as intraluminal shunts for aortic resection and grafting in animals. Johnson, Kirby, and Lehr,[57] and Lam and Aram[28] applied this principle in excising thoracic aneurysms while the aorta was clamped for 6 to 8 minutes and 15 to 24 minutes, respectively. The latter's patient had residual weakness in the lower extremities and a temporary neurogenic bladder, postoperatively.

The second method employs the use of lateral or exclusion shunts whereby the blood flow is never completely interrupted even though the pathologic section of the aorta is completely isolated eventually. Carrel[47] was again a pioneer in attempting the aorta-aortic shunts and shunts from left ventricle to the aorta experimentally. His results were poor. Freeman and Leeds[71] were able to resect aneurysms of the abdominal aorta after shunting the blood flow by way of a splenic artery-internal iliac artery anastomosis. Clatworthy and Varco,[58] and Clatworthy, Sako, Chisholm, and Culmer[59] showed marked improvement in mortality and morbidity rates as compared to those in controls when siliconized polyethylene tubes with a maximal diameter of 0.085 inch were used as external shunts experimentally. Schafer and Hardin,[60] and Hardin, Batchelder, and Schafer[61] similarly had good results in animal experiments. The same authors,[60] however, had two fatalities on attempting to resect an aneurysm of the aortic arch and one of the abdominal aorta, respectively. The method was successful in treating a vena cava–abdominal aorta aneurysm. These authors noted that partial occlusion of the inferior vena cava limited the proximal hypertension and cardiac load by limiting the cardiac input. Izant, Hubay and Holden[62, 63] were able to shunt 80 per cent of the aortic flow through a large polyethylene tube experimentally. The shunt was fixed to the aorta by two double-lumen, double-flanged, stainless steel tubes. Mahorner and Spencer[64] used the homograft itself as an external shunt in treating an aneurysm of the innominate artery. The aneurysm was ligated proximally and distally at the first procedure as well and excised at a later date. Cross, Kay, and Jones,[65] and Donovan[66] have reported experimental results in shunting the left ventricle by way of the auricle to the aorta and the pulmonary conus to the pulmonary artery by way of Tygon tubing with good results suggesting its use for surgery on the valves and proximal great vessels. Stranahan, Alley, Sewell, and Kausel[67] presented a method of shunting any portion of the aorta, including the arch, with the use of a multilimbed Tygon tubing fixed to the proximal and distal aorta with heterografts and to the innominate artery with a cannula, the patient died in the operating room from hemorrhage due to a concomitant pneumonectomy. They successfully excised an aneurysm of the descending thoracic aorta in another case within 3 hours and 20 minutes using a two-limbed shunt. Satinsky, Neptune, and Alai[68] suggested a method of shunting the arch of the aorta with the homograft that is to be its replacement.

As a corollary to the use of external shunt, it has been shown experimentally that the flow of blood through these external shunts can be increased[69] if the shunt is passed through a pump or maintained[0] if the heart is intentionally fibrillated for intracardiac surgery.

DISCUSSION

There have been numerous reports in the recent literature concerning the excision of coarctation, aneurysm, and obliterative processes of the aorta. In the treatment of all these diseases, the aorta is clamped either tangentially or crosswise, which, in the latter instance, always alters the hemodynamics of the circulatory system. This results in, (1) increased load on the heart and coronary system, (2) hypertension proximal to the clamp, (3) hypotension and ischemia distal to the clamp, and (4) occasional circulatory collapse when the clamp is removed.

The several methods of minimizing these sequelae include partial inferior caval occlusion, intraluminal and extraluminal shunts, and hypothermia. When considering the type of procedure to be employed, the type of pathology to be removed and its site are of prime importance.

Obliterative disease usually occurs in the distal aorta and collaterals are well developed, thereby alleviating the need for rapid surgery, shunts, and hypothermia. Here the main problem encompasses the adequacy of the anastomosis between the homograft or plastic prosthesis and the host vessels which are usually markedly sclerotic and may have been further weakened by thromboendarterectomy.[75] Collaterals are also well developed in coarctation of the aorta but, since the occlusion is in the proximal aorta, the problem is greater especially when the coarctation is long or associated with poststenotic dilatation which necessitates a homograft with the additional time needed for two anastomoses rather than a more simple resection and an end to end repair. Accordingly, Gross,[37] Crafoord,[36] Stephens and Grimes,[33] and Glenn and O'Sullivan[31] have all reported fatalities on attempting to treat coarctations. Neurological sequelae have been reported also.

Aneurysms differ from coarctations and obliterative diseases in not causing a comparable collateral arterial system. The problem of anastomosis of the graft to the host aorta in the presence of atherosclerosis, after resection of an aneurysm, is similar to that in obliterative disease and has resulted in fatalities.[32, 30]

Saccular aneurysms are usually found above the renal arteries and are generally due to syphilis. If the sac can be mobilized, restorative endoaneu-

but clamping just proximal to the left carotid artery had to be discontinued after 7 minutes because of hypotension and cardiac irregularity. Further experimental evidence[41-45] revealed that hypothermia lengthened the time that the aorta may be totally occluded but the incidence of complications remained considerable. Investigators have attempted to preserve normal dynamics of circulation in order to avoid the shortcomings of the previously described methods of treatment. This has been accomplished in two ways. In the first, a graft or prosthesis is threaded over a rigid tube that serves as a temporary intraluminal shunt. As early as 1894, Abbe,[46] and Carrel,[47-50] in 1910, pioneered this method when they placed tubes of paraffined rubber, glass, aluminum, and gold plate into major arteries of experimental animals. These tubes functioned as temporary conduits even though they eventually thrombosed. Lately, Hufnagel[51] and others[52-56] successfully used methyl methacrylate, polyethylene, and siliconized stainless steel tubes as intraluminal shunts for aortic resection and grafting in animals. Johnson, Kirby, and Lehr,[57] and Lam and Aram[28] applied this principle in excising thoracic aneurysms while the aorta was clamped for 6 to 8 minutes and 15 to 24 minutes, respectively. The latter's patient had residual weakness in the lower extremities and a temporary neurogenic bladder, postoperatively.

The second method employs the use of lateral or exclusion shunts whereby the blood flow is never completely interrupted even though the pathologic section of the aorta is completely isolated eventually. Carrel[47] was again a pioneer in attempting the aorta-aortic shunts and shunts from left ventricle to the aorta experimentally. His results were poor. Freeman and Leeds[71] were able to resect aneurysms of the abdominal aorta after shunting the blood flow by way of a splenic artery-internal iliac artery anastomosis. Clatworthy and Varco,[58] and Clatworthy, Sako, Chisholm, and Culmer[59] showed marked improvement in mortality and morbidity rates as compared to those in controls when siliconized polyethylene tubes with a maximal diameter of 0.085 inch were used as external shunts experimentally. Schafer and Hardin,[60] and Hardin, Batchelder, and Schafer[61] similarly had good results in animal experiments. The same authors,[60] however, had two fatalities on attempting to resect an aneurysm of the aortic arch and one of the abdominal aorta, respectively. The method was successful in treating a vena cava–abdominal aorta aneurysm. These authors noted that partial occlusion of the inferior vena cava limited the proximal hypertension and cardiac load by limiting the cardiac input. Izant, Hubay and Holden[62, 63] were able to shunt 80 per cent of the aortic flow through a large polyethylene tube experimentally. The shunt was fixed to the aorta by two double-lumen, double-flanged, stainless steel tubes. Mahorner and Spencer[64] used the homograft itself as an external shunt in treating an aneurysm of the innominate artery. The aneurysm was ligated proximally and distally at the first procedure as well and excised at a later date. Cross, Kay, and Jones,[65] and Donovan[66] have reported experimental results in shunting the left ventricle by way of the auricle to the aorta and the pulmonary conus to the pulmonary artery by way of Tygon tubing with good results suggesting its use for surgery on the valves and proximal great vessels. Stranahan, Alley, Sewell, and Kausel[67] presented a method of shunting any portion of the aorta, including the arch, with the use of a multilimbed Tygon tubing fixed to the proximal and distal aorta with heterografts and to the innominate artery with a cannula, the patient died in the operating room from hemorrhage due to a concomitant pneumonectomy They successfully excised an aneurysm of the descending thoracic aorta in another case within 3 hours and 20 minutes using a two limbed shunt Satinsky, Neptune, and Alai[68] suggested a method of shunting the arch of the aorta with the homograft that is to be its replacement

As a corollary to the use of external shunt, it has been shown experimentally that the flow of blood through these external shunts can be increased[69] if the shunt is passed through a pump or maintained[70] if the heart is intentionally fibrillated for intracardiac surgery

DISCUSSION

There have been numerous reports in the recent literature concerning the excision of coarctation, aneurysm, and obliterative processes of the aorta In the treatment of all these diseases the aorta is clamped either tangentially or crosswise, which, in the latter instance, always alters the hemodynamics of the circulatory system This results in, (1) increased load on the heart and coronary system, (2) hypertension proximal to the clamp, (3) hypotension and ischemia distal to the clamp, and (4) occasional circulatory collapse when the clamp is removed

The several methods of minimizing these sequelae include partial inferior caval occlusion, intraluminal and extraluminal shunts, and hypothermia When considering the type of procedure to be employed, the type of pathology to be removed and its site are of prime importance

Obliterative disease usually occurs in the distal aorta and collaterals are well developed, thereby alleviating the need for rapid surgery, shunts and hypothermia Here the main problem encompasses the adequacy of the anastomosis between the homograft or plastic prosthesis and the host vessels which are usually markedly sclerotic and may have been further weakened by thrombo endarterectomy[71] Collaterals are also well developed in coarctation of the aorta but, since the occlusion is in the proximal aorta, the problem is greater especially when the coarctation is long or associated with poststenotic dilatation which necessitates a homograft with the additional time needed for two anastomoses rather than a more simple resection and an end to end repair Accordingly, Gross,[37] Crafoord[36] Stephens and Grimes,[33] and Glenn and O'Sullivan[31] have all reported fatalities on attempting to treat coarctations Neurological sequelae have been reported also

Aneurysms differ from coarctations and obliterative diseases in not causing a comparable collateral arterial system The problem of anastomosis of the graft to the host aorta in the presence of atherosclerosis, after resection of an aneurysm, is similar to that in obliterative disease and has resulted in fatalities[38, 39]

Saccular aneurysms are usually found above the renal arteries and are generally due to syphilis If the sac can be mobilized, restorative endo aneu

but clamping just proximal to the left carotid artery had to be discontinued after 7 minutes because of hypotension and cardiac irregularity. Further experimental evidence[41-45] revealed that hypothermia lengthened the time that the aorta may be totally occluded but the incidence of complications remained considerable. Investigators have attempted to preserve normal dynamics of circulation in order to avoid the shortcomings of the previously described methods of treatment. This has been accomplished in two ways. In the first, a graft or prosthesis is threaded over a rigid tube that serves as a temporary intraluminal shunt. As early as 1894, Abbe,[46] and Carrel,[47-50] in 1910, pioneered this method when they placed tubes of paraffined rubber, glass, aluminum, and gold plate into major arteries of experimental animals. These tubes functioned as temporary conduits even though they eventually thrombosed. Lately, Hufnagel[51] and others[52-56] successfully used methyl methacrylate, polyethylene, and siliconized stainless steel tubes as intraluminal shunts for aortic resection and grafting in animals. Johnson, Kirby, and Lehr,[57] and Lam and Aram[28] applied this principle in excising thoracic aneurysms while the aorta was clamped for 6 to 8 minutes and 15 to 24 minutes, respectively. The latter's patient had residual weakness in the lower extremities and a temporary neurogenic bladder, postoperatively.

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shunted, whether by special clamps,[62] [63] heterografts,[6] or fabrics of adequate length and diameter,[91] indicate that at least the blood pressure changes and distal ischemia may be successfully avoided

SUMMARY

1 Resection of lesions high in the thoracic aorta should be done in a manner to avoid cerebral hypertension, left ventricular strain, and ischemia of distal vital organs

2 External shunts of large diameter will avoid these complications and may consist of heterologous grafts, tubes, or fabrics

3 In a clinical case a heterologous shunt (pig's aorta) was used between the ascending and abdominal aorta while four aneurysms in the ascending and descending aorta were successfuly removed A homograft was used to replace the descending aorta

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DISCUSSION

DR. WILLIAM H. SEWELL, Bethesda, Md.—The technique of using a heterologous arterial shunt can also be used when one desires to divide the aorta proximal to the innominate artery. It requires at least a three-limb rather than a two-limb shunt.

(Movie) This is a dog operation which was done together with a group from Albany. The shunts we use are the brachiocephalic artery from about 1,200-pound steers. The bovine species have only one major branch coming from the aortic arch and this gives rise to both subclavians and both carotids. The brachiocephalic artery is removed from the donor animal very near the arch. One of the branches is sutured to the ascending aorta, and the second to the descending. The third can go to the brachiocephalic artery in the case of the dog which has only two branches from its arch. In the case of the human, the remaining two can be used for the innominate and the left carotid arteries.

Here we see the shunt in action. The limb is sutured to the ascending aorta of the dog; this one is to the descending aorta, and here is a polyethylene cannula connecting into the brachiocephalic artery of the dog. These bovine shunts, like the pig ones, pulsate nicely, and as Dr. Alley has previously reported, the major advantage is the adsorption of the systolic thrust from the proximal aorta. Blood can be stored in the shunt during systole and returned during diastole. The shunt in this case has been sutured to the aorta by end-to-side anastomosis exactly as in the film you just saw.

These vessels are about the size of one's middle finger, and we believe they are large enough both for the dog and for human beings. If a larger one is desired, a larger donor could be obtained.

The procedure is difficult and rather dangerous, but we believe it has a very definite place in surgery of this part of the aorta. The extra time required for implanting and removing the shunt is often repaid by the greater safety provided.

DR. FREDERICK S. CROSS, Cleveland, Ohio.—Several criticisms may be directed at the use of artificial shunts to by-pass segments of thoracic aorta during aortic resection. Plastic shunts of sufficient caliber are difficult to insert and are rigid, making them unwieldy and in the way during surgery. In the case of homologous or heterologous blood vessel shunts, at least two extra anastomoses are required, and there is the necessity of repairing the vessels following the removal of the shunts.

(slide) In order to get around some of these objections through the use of small-caliber flexible plastic tubing, we have placed a Sigmamotor pump in the circuit to obtain the flow rates desired. In by-passing the descending thoracic aorta, blood is picked up from the left subclavian artery as shown and returned to the descending aorta or into the femoral artery.

(slide) The next slide illustrates how the pressures can be regulated above and below the by-passed aortic segment. By increasing the pump rate it is seen that the aortic arch pressures are lowered and the abdominal aortic pressures are raised. It is possible, then, to select and maintain the pressure one wants to carry in the aortic arch region and to avoid extreme degrees of hypertension in this area.

(slide) The most difficult problem associated with the resection of the aorta is that of resecting the arch region. A year ago we presented work before this Association utilizing a rigid plastic shunt from the left ventricle to the aorta. It works fairly well but, unfortunately, is not the final answer because in order to get adequate flow rates and to prevent hypertension in the left ventricle it is necessary to use a fairly large caliber tube (at least one-half of the diameter of the ascending aorta).

(slide) A more satisfactory technique is to utilize the Sigmamotor pump with small tubing, taking blood directly from the left ventricle and returning it to the innominate and carotid arteries and to the descending aorta. In this manner, the arch can be completely by-passed. It is probably simpler, however, to return the blood only to the descending aorta or femoral artery, suturing the graft in a retrograde manner, first making an end-to-side anastomosis between the graft and descending aorta, then between the graft and the vessels in the arch, and finally between the graft and the ascending aorta In this manner, the ascending aorta need be clamped only during the final anastomosis and blood flow to the vital centers is maintained throughout the insertion of the graft

DR HENRY T BAHNSON, Baltimore, Md —I should like to compliment Dr Chamberlain and his group for a beautiful combination of surgical skill, modern cinematographic technique, comparative anatomy, and the transportation system of the Eastern seaboard

Any procedure, however, which requires ten hours at the hands of Dr Chamberlain, I am sure will require considerably longer than that at mine and at the hands of many others, and I would like to show simply a technique used in a patient which solved some of our overtime hours The patient had an aneurysm on the aortic arch The sizable aneurysm and clot can be seen as well as the lumen filled with contrast material This specimen is demonstrated here in the best Houston fashion

(slide) This shows the operative procedure We simply put our definitive shunt in, as described by Mahorner and Spencer, instead of using a heterograft This was anastomosed to the proximal aorta and the distal end was anastomosed to the distal aorta We initially planned to anastomose the various vessels of the arch to the head vessels, but found after the shunt was in place and after dissection of the aneurysm had begun, that we were able to place a clamp across the lower side of the aortic arch and preserve a tongue of aorta from which the cerebral and brachial vessels arose The obvious disadvantage of this type of procedure is that more graft tissue is left in place than is normally required, in fact, this has led to considerable chiding by one of my aneurysmal friends concerning a patient in whom a thoracic aneurysm was so large that we had to sew two thoracic aortic grafts together to bridge it The extra graft, however, that was required in this individual in order to get around the aneurysm has caused no embarrassment to the patient and he is well now some two months following the procedure

DR CLARENCE CRAFOORD, Stockholm, Sweden —Dr Chamberlain raised one point of interest and importance, that was the release of the clamps in cross clamping the aorta In close to 200 patients with aortic resections, we have not experienced any complications in spite of the fact that we have never taken any precaution whatsoever to release the clamps slowly

We have, also, between 30 and 40 cases of patent ductus where the cross clamps below and above the ductus were in for periods of up to between 15 and 30 minutes and, even in those cases, we have not seen any blood pressure variations of any consequence after quick release of the clamps I have had no experience with cross clamping of the aorta when there is no collateral circulation for longer than a period of up to half an hour The reports that have come from this country, and from other places as well, on the danger in releasing cross clamps very rapidly, I believe must be due to something other than the cross clamping itself I believe that the deleterious effect ascribed to the quick release of the clamps is due to the fact that the patient has not been under good anesthesia and good ventilation during the time of operation The type of ventilation that I see in many places is not often adequate If arterial blood samples had been taken from patients in whom circulatory complications arose after rapid release of cross clamps, I am certain one would have found astonishingly high carbon dioxide tensions and acidosis, and probably also low oxygen tension such as we have been able to show when we have experimentally used the type of ventilation most often used, something that Dr Gibbon and his group now have shown so beautifully

DR I D DODRILL, Detroit, Mich —If one is to use a by pass for the thoracic aorta, one will occasionally encounter an aneurysm which is low down in the thoracic aorta near the diaphragm and, in order to by pass it, one must go lower in the arterial system.

(slide) This is a way of by passing the thoracic aorta in which one removes the blood from the left subclavian artery, passes it out through the pump, and puts it back in the femoral artery in a retrograde manner A tube by pass from the subclavian to the femoral arteries had been done before This is not original with me We have carried out experimental work in which we measured the pressure in the lower segment and the upper seg

DISCUSSION

DR. WILLIAM H. SEWELL, Bethesda, Md.—The technique of using a heterologous arterial shunt can also be used when one desires to divide the aorta proximal to the innominate artery. It requires at least a three-limb rather than a two-limb shunt.

(Movie) This is a dog operation which was done together with a group from Albany. The shunts we use are the brachiocephalic artery from about 1,200-pound steers. The bovine species have only one major branch coming from the aortic arch and this gives rise to both subclavians and both carotids. The brachiocephalic artery is removed from the donor animal very near the arch. One of the branches is sutured to the ascending aorta, and the second to the descending. The third can go to the brachiocephalic artery in the case of the dog which has only two branches from its arch. In the case of the human, the remaining two can be used for the innominate and the left carotid arteries.

Here we see the shunt in action. The limb is sutured to the ascending aorta of the dog; this one is to the descending aorta, and here is a polyethylene cannula connecting into the brachiocephalic artery of the dog. These bovine shunts, like the pig ones, pulsate nicely, and as Dr. Alley has previously reported, the major advantage is the adsorption of the systolic thrust from the proximal aorta. Blood can be stored in the shunt during systole and returned during diastole. The shunt in this case has been sutured to the aorta by end-to-side anastomosis exactly as in the film you just saw.

These vessels are about the size of one's middle finger, and we believe they are large enough both for the dog and for human beings. If a larger one is desired, a larger donor could be obtained.

The procedure is difficult and rather dangerous, but we believe it has a very definite place in surgery of this part of the aorta. The extra time required for implanting and removing the shunt is often repaid by the greater safety provided.

DR. FREDERICK S. CROSS, Cleveland, Ohio.—Several criticisms may be directed at the use of artificial shunts to by-pass segments of thoracic aorta during aortic resection. Plastic shunts of sufficient caliber are difficult to insert and are rigid, making them unwieldy and in the way during surgery. In the case of homologous or heterologous blood vessel shunts, at least two extra anastomoses are required, and there is the necessity of repairing the vessels following the removal of the shunts.

(slide) In order to get around some of these objections through the use of small-caliber flexible plastic tubing, we have placed a Sigmamotor pump in the circuit to obtain the flow rates desired. In by-passing the descending thoracic aorta, blood is picked up from the left subclavian artery as shown and returned to the descending aorta or into the femoral artery.

(slide) The next slide illustrates how the pressures can be regulated above and below the by-passed aortic segment. By increasing the pump rate it is seen that the aortic arch pressures are lowered and the abdominal aortic pressures are raised. It is possible, then, to select and maintain the pressure one wants to carry in the aortic arch region and to avoid extreme degrees of hypertension in this area.

(slide) The most difficult problem associated with the resection of the aorta is that of resecting the arch region. A year ago we presented work before this Association utilizing a rigid plastic shunt from the left ventricle to the aorta. It works fairly well but, unfortunately, is not the final answer because in order to get adequate flow rates and to prevent hypertension in the left ventricle it is necessary to use a fairly large caliber tube (at least one-half of the diameter of the ascending aorta).

(slide) A more satisfactory technique is to utilize the Sigmamotor pump with small tubing, taking blood directly from the left ventricle and returning it to the innominate and carotid arteries and to the descending aorta. In this manner, the arch can be completely by-passed. It is probably simpler, however, to return the blood only to the descending aorta or femoral artery, suturing the graft in a retrograde manner, first making an end-to-side anastomosis between the graft and descending aorta, then between the graft and the vessels in the arch, and finally between the graft and the ascending aorta In this manner, the ascending aorta need be clamped only during the final anastomosis and blood flow to the vital centers is maintained throughout the insertion of the graft

DR HENRY T BAHNSON, Baltimore, Md —I should like to compliment Dr Chamberlain and his group for a beautiful combination of surgical skill modern cinematographic technique, comparative anatomy, and the transportation system of the Eastern seaboard

Any procedure, however, which requires ten hours at the hands of Dr Chamberlain, I am sure will require considerably longer than that at mine and at the hands of many others, and I would like to show simply a technique used in a patient which solved some of our overtime hours The patient had an aneurysm on the aortic arch The sizable aneurysm and clot can be seen as well as the lumen filled with contrast material This specimen is demonstrated here in the best Houston fashion

(slide) This shows the operative procedure We simply put our definitive shunt in, as described by Mahorner and Spencer, instead of using a heterograft This was anastomosed to the proximal aorta and the distal end was anastomosed to the distal aorta We initially planned to anastomose the various vessels of the arch to the head vessels, but found after the shunt was in place and after dissection of the aneurysm had begun, that we were able to place a clamp across the lower side of the aortic arch and preserve a tongue of aorta from which the cerebral and brachial vessels arose The obvious disadvantage of this type of procedure is that more graft tissue is left in place than is normally required, in fact, this has led to considerable chiding by one of my aneurysmal friends concerning a patient in whom a thoracic aneurysm was so large that we had to sew two thoracic aortic grafts together to bridge it The extra graft, however, that was required in this individual in order to get around the aneurysm has caused no embarrassment to the patient and he is well now some two months following the procedure

DR CLARENCE CRAFOORD, Stockholm, Sweden —Dr Chamberlain raised one point of interest and importance, that was the release of the clamps in cross clamping the aorta In close to 200 patients with aortic resections, we have not experienced any complications in spite of the fact that we have never taken any precaution whatsoever to release the clamps slowly

We have, also, between 30 and 40 cases of patent ductus where the cross clamps below and above the ductus were in for periods of up to between 15 and 30 minutes and, even in those cases, we have not seen any blood pressure variations of any consequence after quick release of the clamps I have had no experience with cross clamping of the aorta when there is no collateral circulation for longer than a period of up to half an hour The reports that have come from this country, and from other places as well, on the danger in releasing cross clamps very rapidly, I believe must be due to something other than the cross clamping itself I believe that the deleterious effect ascribed to the quick release of the clamps is due to the fact that the patient has not been under good anesthesia and good ventilation during the time of operation The type of ventilation that I see in many places is not often adequate If arterial blood samples had been taken from patients in whom circulatory complications arose after rapid release of cross clamps, I am certain one would have found astonishingly high carbon dioxide tensions and acidosis, and probably also low oxygen tension such as we have been able to show when we have experimentally used the type of ventilation most often used, something that Dr Gibbon and his group now have shown so beautifully

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(slide) This is a way of by passing the thoracic aorta in which one removes the blood from the left subclavian artery, passes it out through the pump, and puts it back in the femoral artery in a retrograde manner A tube by pass from the subclavian to the femoral arteries had been done before This is not original with me We have carried out experimental work in which we measured the pressure in the lower segment and the upper segment, both, with a simple tube by-pass and with a pump exerting some force on the blood of the lower segment. With a simple tube by-pass, with a cannula as large as will go in the subclavian, and as large as will go in the femoral, the pressures are very low in the lower aortic segment and we found that a pressure of 40 to 45 mm. Hg was about as high as it would go. If one exerts pumping action on the blood, one can raise the pressure in the lower segment to that of the upper segment so that hemodynamically the animal is then in a good physiologic state. There are obvious disadvantages to this method; one is, that if one pumps blood for very long one must use some heparin. This is not a serious objection. The more serious objection is that in most patients, especially if one is dealing with a sclerotic aneurysm, there is also plaque formation in the femoral artery and one would not wish to do this type of procedure. However, if the femoral artery is reasonably healthy and does not contain plaques, one can cannulate it in a retrograde manner and resuture it without serious difficulty.

DR. J. D. FRYFOGLE, Detroit, Mich.—I would like to tell you about a method of arterial anastomosis that is new, is safe, is simple, and avoids complicated shunts and hypothermia.

(slide) We are looking at twin pieces of stainless steel that are held together by a simple polythene plastic ring. With the use of these little vessel couples, as we call them, we are able to place anywhere in the arterial tree a free graft with a minimum of time, a minimum of effort both for the patient and for the surgeon, and to establish a good flow of blood. This is also pertinent to the subject.

(slide) This is how it is done. The couple looks like the upper picture and is held together by the plastic ring. You pull the graft to be placed through the little steel device, cuff it as you would a Blakemore tube and tie it at the end if you desire to hold it in place. Merely insert it as you would the Blakemore tube and put a single suture around it. Having made the suture tie at this point, there is then a free flow of blood. It takes approximately 45 seconds, with a team that is practiced, to re-establish circulation through a single couple. The surgeon can then sew at his leisure. It is a little difficult to demonstrate and I wish I had time to present the movies, but you sew in this groove, which can be readily felt through two thicknesses of vessel wall, and it is a simple suture that gives pretty good closure. It may be necessary to put in a few interrupted sutures when you are through. At the conclusion of the suture, cut the tie, cut the plastic ring, the whole couple comes apart and an intima-to-intima sutured anastomosis is complete.

The first couple was devised by Dr. Walter Stenborg in 1951 and was an attempt to bridge arterial grafts with venous segments. It did not work because of suturing difficulty. When arterial transplants became available, I redesigned the couple, made raises in it instead of grooves and it has worked satisfactorily in our laboratory.

(slide) These are little things you can do. You can take these little segments of the aortic arch and have blood flowing through a coarctation specimen in this length of time. You can take the typical Leriche syndrome and have blood flowing in three minutes and five seconds. This is a more complicated situation and I have found out many things while attempting this anastomosis. There are different sizes and takeoffs in the renal vessels, and it is very difficult to get an approach that is satisfactory in order to make this anastomosis.

(slide) Here is the subject we are talking about and this is the time it takes to get blood flowing to the brain. Actually, all the couples are not inserted at one time. You make this one (aortic) and this one (innominate) and put a clamp across the graft so that the blood is flowing somewhere under three minutes. Move over and put the next couple (carotid) in and there is a total blood supply to the brain under this stated time because you still allow 45 seconds for this (distal aorta).

(slide) This is the most important slide of the presentation because it covers the three vantage points you must see. When you have an aneurysmal dilatation do not fuss with it; you do not want to work over a bump. Once the clamp is put down you can split the sac lengthwise and cut the vessel ends, leaving room for the couples. Be very careful to cut about two-thirds of the way around. This immediately accomplishes three things. It gets rid of the mass, there is room to work and to put the graft in place. Second, it does not allow the vessels to retract away from you, so there is no trouble measuring the length of the graft, the graft is already prepared and everything is ready to move by the time the initial clamping is made This fits in with a simple tie around here and the blood is flowing somewhere (in this particular case) in a little over two minutes

(slide) This is a dog in which I put a graft some three months ago but I was worried because I had never seen suture lines after that length of time This is a suture line which is beautifully epithelized We put a third aorta in this dog and he is still well and happy

DR CHAMBERLAIN (Closing) I feel a little bit like the Southern surgeon who reported 500 cases of thyroid disease before one of his societies and a second surgeon reported 1,000, and a third surgeon reported 2 000 In his closing discussion he said, "Well, I'm glad to have provided the can opener for this picnic!"

I would like to add one thing Dr Crafoord referred to sudden removal of the clamp in coarctations and, in my opening statement, I mentioned that there was collateral circulation in these cases and I feel quite sure it is relatively safe to release the clamp suddenly in accordance with his recommendation of several years ago In young normal dogs, however there is little collateral circulation In them if we clamp the aorta just distal to the dog's left innominate artery for an hour and then suddenly release it the pressure falls dramatically to almost zero and comes back very slowly, so that sometimes it is half an hour before the blood pressure returns to about 80 or 90 In old dogs, if we clamp the aortas in a similar way and then suddenly release the clamp, we have some of them go immediately into arrest or ventricular fibrillation and die I do not know that the explanation I have given you is the entire explanation and, with Professor Crafoord I must admit that there are probably other factors, but I believe in the absence of collateral circulation sudden release of the clamp is a hazardous procedure

Dr Fryfogle has a very nice solution to this problem and I am looking forward to following his work If he can clamp the ascending aorta, especially in old patients for three or four minutes successfully then his solution is superior to the shunt technique Certainly clamping the ascending aorta and dividing it as we have done in the dog laboratory when we had another objective in mind, was disastrous in a huge majority of the dogs in whom the circulation was stopped for more than four to six minutes Anyone who has had occasion to clamp the ascending aorta knows the dramatic response of the left ventricle to the sudden terrific head pressure

I wish to thank all the discussers for their contributions

Table II. Clinical Data and Hemodynamic Variables Studied Before, During and After

E	AGE (YEARS)	SEX	BEFORE OPERATION				DURING OPERATION PRESSURE (MM. HG)			
			PRESSURE (MM. HG)		NET SHUNT L-TO-R PER CENT PULMONARY FLOW	SHUNT R-TO-L DEMONSTRATED‖	BEFORE CLOSURE		AFTER CLOSURE	
			PULMONARY ARTERY	RADIAL ARTERY			PULMONARY ARTERY	RADIAL ARTERY	PULMONARY ARTERY	RADIAL ARTERY
	13	F	82/42	102/45	70		112/72	120/78	82/46	148/98
	14	F	94/63	111/54	65	+++	100/75	102/65	50/35¶	93/70¶
	24	F	65/41	110/57	64					
	46	F	62/38	176/58	60					
	5	F	68/42	97/47	59					
	59	F	62/30	134/63	57					
	12	M	106/68	168/63	57	+++	113/73	163/94	120/86	158/82
*	5	M	90/53	121/52	53	+++				
	42	F	67/34	272/119	40					
	28	F	82/53	132/63	37					
	21	M	105/78	110/70	37	+++				
	59	M	84/47†	90/60	36	+++				
	7	F	87/50	111/65	34					
	25	F	112/66	160/74	32					
	27	M	61/41	182/70	27					
	2½ mo.	M	69/37†	73/35	23		75/45	80/40§	60/35	108/55§
	16 mo.	F	118/69†	112/65	10		95/40	112/47	71/22	135/72
	35	F	109/73	115/74	8	+++				
	17	F	130/80	133/75	5	+++				
	35	M	130/85	133/80	5	+++				
	7½	M	106/76†	100/69	<5	+++	109/73	92/66	131/83	79/49
	3	M	90/60†	120/70§						
§,	4¾	M	86/70†	95/57		+++				
›	19 mo.	F	131/70†	108/62	0	+++				
	9	M	71/62†	94/70						
	45	F	143/78	140/69	0	+++				
	21	F	101/62	105/67	0	+++				
	7 mo.	F	74/43†	107/32			85/60	90/70	67/43	110/85
	24	F	104/62	127/68	0	+++				
	4	M	89/10†‡							

*Aortic-pulmonary septal defect.

†Studied under anesthesia.

‡Right ventricular pressure.

§Aortic pressure.

‖Right-to-left shunts undoubtedly were present at some time in other patients but were either not sent at catheterization or were not demonstrated.

¶Pressures recorded after profuse hemorrhage.

the varying opinions that have been expressed. Thus, it has been stated that the presence of pulmonary hypertension is a contraindication to operation,[21] that patients who exhibit a right-to-left shunt should not undergo operation,[42-44] and that operation should be undertaken in the presence of a right-to-left shunt only if it is less than the left-to-right shunt.[22, 45] It is hoped that the following analysis of data on 30 patients treated surgically at the Mayo Clinic will shed further light on this problem.

Operation in 30 Cases of Aortic Pulmonary Communication With Pulmonary Hypertensio

After Operation Pressure (mm. Hg)			Clinical Data		
MONTHS AFTER OPERATION	PULMONARY ARTERY	RADIAL ARTERY	ASSOCIATED DEFECT	INTERVAL (YEARS)	RESULT
8	45/15	110/60		73	Well
				¾	Partial paraplegia fro cross clamping aorta cardiovascular syste excellent
4	36/13	113/70	Ventricular septal defect	1⅓	Well
				3¾	Minimal dyspnea
					Lost to follow up
				1⅓	Heart failure controll with digitalis
4	51/13	134/75	Coarctation	1	Well
11	35/8	83/61		1½	Well
4½	35/16	208/117		1⅓	Well
7½	38/13	118/74		7⁄12	Well
10	54/16	129/73		1	Minimal dyspnea
				4½	Well
				1	Well
12	89/48	160/74	Atrial septal defect	1¼	Well, recanalized
5¼	33/10		Coarctation	4¼	Well
				⅔	Well
				1⅓	Well
					Fatal hemorrhage
			Ventricular septal defect	2¾	Well
7⅙	138/80	179/94		2⅓	Recanalized, died
				½	Died
17	21/7†	89/62		3⅓	Well
			Coarctation		Died immediately posto
			Coarctation	1⅓	Well
24½	60/25†	110/70		2¾	Well, recanalized
				8 days	Died suddenly
					Fatal hemorrhage
				1⅙	Well
					Died immediately posto
			Atrial septal defect	4½	Well

ANALYSIS OF CASES

General Considerations.—Two hundred and seventy one patients with patent ductus arteriosus and 1 patient with an aortic pulmonary septal defect have undergone operation at the Mayo Clinic as of April 1, 1955. Of the 272 patients, 45 had an associated pulmonary hypertension. In 15 cases the presence of pulmonary hypertension was suggested by clinical and operative findings. In 30 cases, cardiac catheterization carried out before operation demonstrated a systolic pulmonary arterial pressure in excess of 60 mm. Hg. This discussion will be restricted to an analysis of these 30 cases. Some patients with patent ductus arteriosus, pulmonary hypertension, and right to left shunt seen at the Clinic have not been subjected to operation and are not included in this analysis.

Certain clinical data and hemodynamic variables measured before, during, and after operation in these 30 patients are summarized in Table II. The patients' ages ranged from 2½ months to 59 years, one half of the patients being less than 15 years of age. Four patients had an associated coarctation of the aorta, 2 had an atrial septal defect, and 2 had a ventricular septal defect. The association of patent ductus arteriosus and coarctation of the aorta may pose a somewhat different hemodynamic problem from that encountered in patients with patent ductus arteriosus alone. For this reason, these 4 cases will not be analyzed in detail but will be presented in another report.[46]

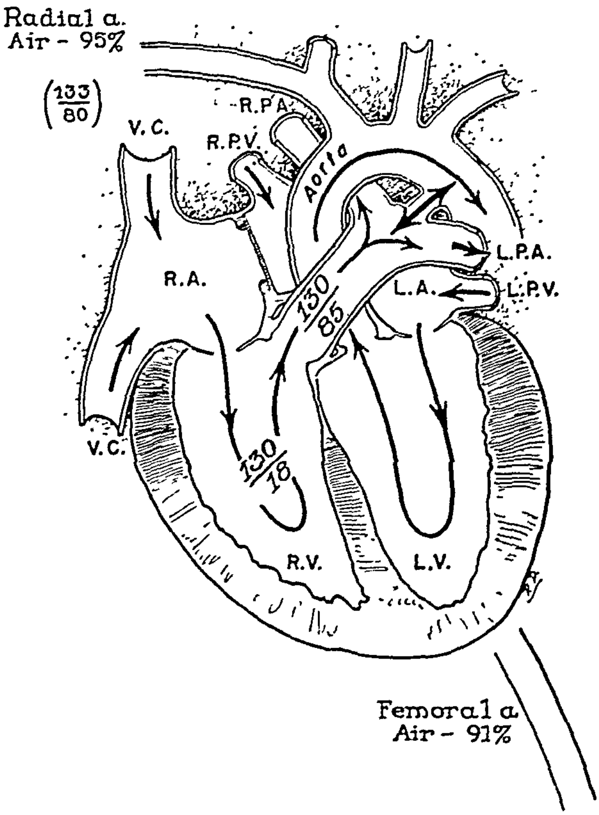

Fig. 1.—Hemodynamic findings in a case of patent ductus arteriosus and pulmonary ertension with a bidirectional shunt (Case 20) Note oxygen saturation of 95 per cent in radial artery and 91 per cent in the femoral artery while the patient was breathing air.

For purposes of convenience the patients are listed in order of net left-to-right shunts of decreasing magnitude. These net left-to-right shunts were calculated on the bases of pulmonary and systemic arterial blood flows. In a few cases, sufficient data were not available to calculate the magnitude of the shunt. Pulmonary arterial pressure varied from a low of 61 mm. of Hg systolic to a high of 143 mm. Hg, there being 15 patients with systolic pulmonary arterial pressures of less than 90 mm. and 15 patients with pressures of 90 mm. or more.

In 14 of these patients a right-to-left shunt was demonstrated. Unquestionably, there were other patients in this series who had a right-to-left shunt

in greater detail later. A seventh patient in whom a reversal of blood flow through the ductus was demonstrated preoperatively was shown by recatheterization seven and one-half months after ligation to have arterialization of the blood in the pulmonary artery, indicating recanalization of the ductus. This patient died two years and four months after operation.

In addition to the occurrence of 7 deaths, certain complications developed in this series of patients with patent ductus arteriosus and pulmonary hypertension. Serious hemorrhage from the ductus in 1 patient necessitated prolonged cross clamping of the aorta. Reversal of blood flow through the ductus was demonstrated preoperatively in this patient. The patient survived operation but was partially paraplegic as a result of the prolonged period of interruption of aortic blood flow. In 3 patients, to 1 of whom reference has already been made, closure of the ductus was carried out by ligation early in the series. At postoperative catheterization, arterialization of the blood in the pulmonary artery was noted, indicating recanalization of the ductus.

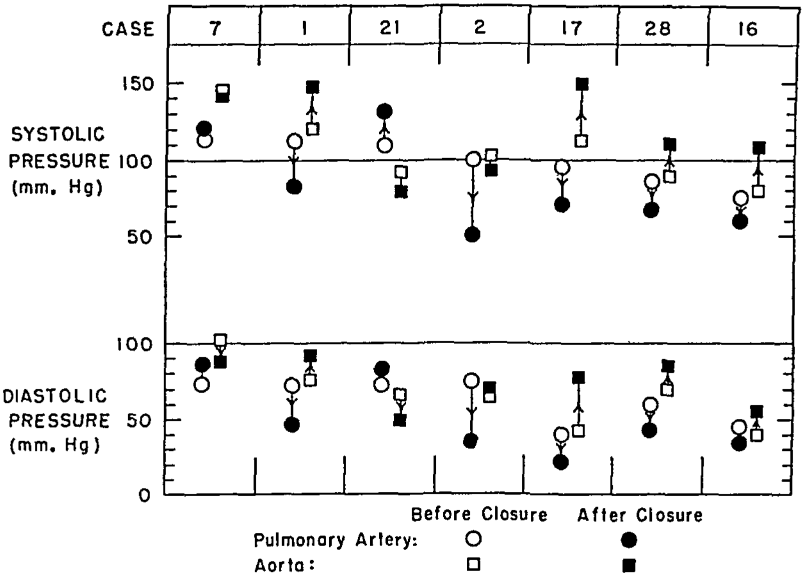

Fig. 2.—Immediate effects of closure of ductus on aortic and pulmonary arterial pres- in 7 cases of patent ductus arteriosus with pulmonary hypertension. The cases are iped in order of decreasing preoperative systolic pressures in the pulmonary artery. Note that in 2 patients (Cases 7 and 21), there was an immediate rise in pulmonary arterial pressure and a fall in aortic pressure and that in the other 5 there was an immediate decrease in pulmonary arterial pressure and a rise in aortic pressure.

One patient, aged 59 years, who had a patent ductus arteriosus and pulmonary hypertension and in whom no right-to-left shunt was demonstrated returned in heart failure six months after division of the ductus. Her symptoms were well controlled by adequate digitalization.

Of the 23 surviving patients, then, 1 remains partially paraplegic but in excellent status as regards the cardiovascular system, 1 has mild heart failure that is well controlled by digitalis, and 1 has been lost to follow-up. Twenty patients are well, including 2 patients in whom there is objective evidence of recanalization of the ductus. In 5 of these 20 patients, a right-to-left shunt was demonstrated preoperatively.

Hemodynamic Results.—Hemodynamic studies are of value in furnishing objective evidence of the patient's response to operative closure of the abnormal aortic-pulmonary communication. Such studies were carried out either during or after operation in 17 patients (Table II). Pulmonary arterial pressure was measured on the operating table before and after occlusion of the ductus in 7 patients (Fig. 2). A fall in pulmonary arterial pressure and a rise in systemic pressure were noted in 5 patients. The significance of these findings will be discussed subsequently.

There were 12 patients whose hearts were recatheterized from four months to more than 2 years after operation. A satisfactory drop in pulmonary arterial pressure was observed in 9 cases. In 3 patients previously referred to, elevated pulmonary arterial pressure persisted in the presence of a recanalized ductus (Cases 14, 20, and 25). In all but 2 of the remaining 8 cases (Cases 7, and 11), pulmonary arterial pressure returned toward normal range. In Cases 7 and 11, there was a drop of about 50 per cent in pulmonary arterial pressure, although a normal level was not reached. In both of these cases a right-to-left shunt was demonstrated preoperatively. Such a shunt, however, does not necessarily preclude a striking drop in pulmonary arterial pressure practically to normal levels following closure of the abnormal communication. Case 8, in which there was an aortic-pulmonary septal defect, is an example, the pulmonary arterial pressure dropping from 90/53 before operation to 35/8 eleven months after closure of the abnormal opening. A detailed report of this case has already been made.[47]

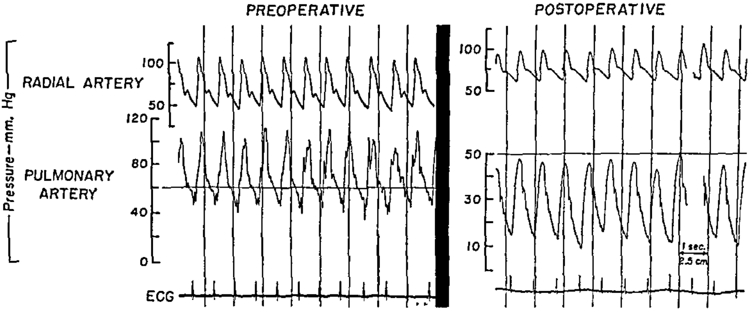

Fig 3.—Sections of photographic record obtained at cardiac catheterization before and eight months after closure of a patent ductus arteriosus in a patient with associated pulmonary hypertension (Case 1). The pulmonary arterial pressure, measured postoperatively, is still slightly elevated but there has been a substantial decrease toward normal levels. The preoperative pulmonary arterial and postoperative radial arterial pressures given in the illustration differ slightly from those in Table I but are not inconsistent; the pressure levels recorded in Table I are representative of the total study.

The most striking drop in pulmonary arterial pressure was demonstrated in Case 22; the details of this case have been reported previously.[48] From a preoperative level of 90/60, the pressure in the pulmonary artery fell to 21/7 seventeen months after division of the ductus. *Portions of the catheterization* records obtained before and after operation in Case 1 are reproduced in Fig. 3. From a preoperative level of 82/42, the pulmonary arterial pressure fell to 45/15 eight months after division of the ductus.

In 6 patients it was possible to calculate preoperative and postoperative total pulmonary arterial and pulmonary arteriolar resistances (Table IV).

TABLE IV. PREOPERATIVE AND POSTOPERATIVE DETERMINATIONS OF BLOOD FLOW AND PULMONARY VASCULAR RESISTANCE IN SIX CASES OF AORTIC-PULMONARY COMMUNICATION WITH PULMONARY HYPERTENSION

CASE	AGE (YEARS)	PREOPERATIVE STUDIES: R-TO-L SHUNT DEMONSTRATED	PREOPERATIVE STUDIES: BLOOD-FLOW INDEX (L./MIN./M.²): PULMONARY	PREOPERATIVE STUDIES: BLOOD-FLOW INDEX (L./MIN./M.²): SYSTEMIC	PREOPERATIVE STUDIES: PULMONARY VASCULAR RESISTANCE* (DYNES SEC. CM.⁻⁵): TPR	PREOPERATIVE STUDIES: PULMONARY VASCULAR RESISTANCE* (DYNES SEC. CM.⁻⁵): PAR	POSTOPERATIVE STUDIES: CARDIAC INDEX (L./MIN. / M.²)	POSTOPERATIVE STUDIES: PULMONARY VASCULAR RESISTANCE* (DYNES SEC. CM.⁻⁵): TPR	POSTOPERATIVE STUDIES: PULMONARY VASCULAR RESISTANCE* (DYNES SEC. CM.⁻⁵): PAR
7	12	+	6.7	3.0	860	620	3.5	569	372
8	5	+	10.7	5.0	630	520	3.3	600	340
9	42		6.8	4.1	860	630	3.9	250	
10	28		5.9	3.7	481	380	5.2	180	128
11	21	+	2.7	1.7	1595		1.8	1060	
15	27		2.6	2.4	1520		3.4	225	

*TPR, total pulmonary arterial resistance; PAR, pulmonary arteriolar resistance.

These data emphasize two factors of importance. Cases 7, 8, and 11 serve to re-emphasize the fact that the presence of a right-to-left shunt does not necessarily indicate a fixed pulmonary vascular resistance and that this resistance may be lowered after closure of the abnormal pulmonary-aortic communication. Lowering of pulmonary vascular resistance occurred in all 3 patients, although normal values were not attained. Case 9 serves to emphasize that age alone is not necessarily a factor in determining the response to operation in patients with this disease. One of the most striking drops in total pulmonary resistance was exhibited by this 42-year-old woman.

Selection of Patients for Operation.—The results among patients in whom only a left-to-right shunt was demonstrated preoperatively, both as regards their immediate response to operation and their long-term progress, leave little question concerning their proper management. All such patients should be advised to have the ductus closed.

TABLE V. HEMODYNAMICS DATA ON THREE PATIENTS WITH PATENT DUCTUS ARTERIOSUS AND PULMONARY HYPERTENSION WHO DIED POSTOPERATIVELY

CASE	AGE (YEARS)	PRESSURE (MM. HG): PULMONARY ARTERY	PRESSURE (MM. HG): RADIAL ARTERY	SHUNTS: L-TO-R PER CENT PULMONARY FLOW	SHUNTS: R-TO-L PER CENT SYSTEMIC FLOW	TPR* (DYNES SEC. CM.⁻⁵)
21	7½	106/76	100/69	<5	40	1660
26	49	143/78	140/69	25	33	1830
29	24	104/62	127/68	21	30	2140

*Total pulmonary arterial resistance.

The group of patients in whom a right to left shunt has been demonstrated poses a more difficult problem. Because it may provide some insight into the possible selection of patients with right to left shunts for operation, data on 3 of 4 such patients who died after operation and on whom sufficient data were available are analyzed in some detail in Table V. In none of the 4 patients was there any obvious cause of death, and post mortem examination, done in 3, failed to add further information. The calculated shunts represent bidirectional shunt components of pulmonary and systemic arterial blood flow and may differ from the *net* left to right shunts in Table II, which represent the percentage by which pulmonary blood flow exceeded systemic blood flow. In all 3 of these patients, the right to left shunt exceeded the left to right shunt. With the possible exception of 1 case (Case 7), which has been reported in detail elsewhere,[49] there are no late survivors after operation in this group of patients in whom a predominantly right to left shunt was demonstrated. The patient in Case 7 also had a coarctation of the aorta, and for this reason may present a somewhat different hemodynamic problem.

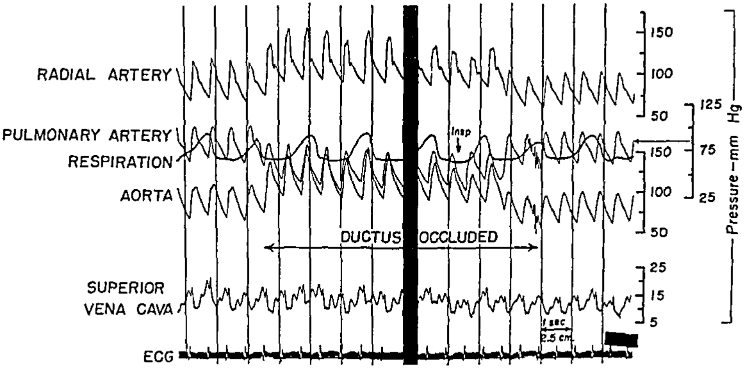

Fig 4.—Sections of photographic record obtained during operation on a patient with patent ductus arteriosus and pulmonary hypertension (Case 1). Note that during temporary occlusion of the ductus arteriosus there was an immediate fall in pulmonary arterial pressure and a rise in aortic and radial arterial pressures. A few seconds later when the ductus was opened these pressures returned approximately to their previous levels.

An undoubtedly related factor, and one which aids the surgeon in making the final decision regarding closure of the ductus, is the hemodynamic response to occlusion of the ductus at the time of operation. The response exhibited to this maneuver in 7 cases is summarized graphically in Fig 2. Values for pulmonary arterial and systemic arterial pressures before and after closure of the ductus are recorded. A fall in pulmonary arterial pressure and a rise in systemic pressure occurred in all but 2 patients. One patient (Case 21) showed a pronounced rise in pulmonary arterial pressure and a fall in systemic pressure when the ductus was occluded. He survived operation but died suddenly six months later. This case has been reported in detail elsewhere.[50] In Case 7

The most striking drop in pulmonary arterial pressure was demonstrated in Case 22; the details of this case have been reported previously.[48] From a preoperative level of 90/60, the pressure in the pulmonary artery fell to 21/7 seventeen months after division of the ductus. Portions of the catheterization records obtained before and after operation in Case 1 are reproduced in Fig. 3. From a preoperative level of 82/42, the pulmonary arterial pressure fell to 45/15 eight months after division of the ductus.

In 6 patients it was possible to calculate preoperative and postoperative total pulmonary arterial and pulmonary arteriolar resistances (Table IV).

TABLE IV. PREOPERATIVE AND POSTOPERATIVE DETERMINATIONS OF BLOOD FLOW AND PULMONARY VASCULAR RESISTANCE IN SIX CASES OF AORTIC-PULMONARY COMMUNICATION WITH PULMONARY HYPERTENSION

CASE	AGE (YEARS)	PREOPERATIVE STUDIES					POSTOPERATIVE STUDIES		
		R-TO-L SHUNT DEMONSTRATED	BLOOD-FLOW INDEX (L./MIN./M.²)		PULMONARY VASCULAR RESISTANCE* (DYNES SEC. CM.⁻⁵)		CARDIAC INDEX (L./MIN./M.²)	PULMONARY VASCULAR RESISTANCE* (DYNES SEC. CM.⁻⁵)	
			PULMONARY	SYSTEMIC	TPR	PAR		TPR	PAR
7	12	+	6.7	3.0	860	620	3.5	569	372
8	5	+	10.7	5.0	630	520	3.3	600	340
9	42		6.8	4.1	860	630	3.9	250	
10	28		5.9	3.7	481	380	5.2	180	128
11	21	+	2.7	1.7	1595		1.8	1060	
15	27		2.6	2.4	1520		3.4	225	

*TPR, total pulmonary arterial resistance; PAR, pulmonary arteriolar resistance.

These data emphasize two factors of importance. Cases 7, 8, and 11 serve to re-emphasize the fact that the presence of a right-to-left shunt does not necessarily indicate a fixed pulmonary vascular resistance and that this resistance may be lowered after closure of the abnormal pulmonary-aortic communication. Lowering of pulmonary vascular resistance occurred in all 3 patients, although normal values were not attained. Case 9 serves to emphasize that age alone is not necessarily a factor in determining the response to operation in patients with this disease. One of the most striking drops in total pulmonary resistance was exhibited by this 42-year-old woman.

Selection of Patients for Operation.—The results among patients in whom only a left-to-right shunt was demonstrated preoperatively, both as regards their immediate response to operation and their long-term progress, leave little question concerning their proper management. All such patients should be advised to have the ductus closed.

TABLE V. HEMODYNAMICS DATA ON THREE PATIENTS WITH PATENT DUCTUS ARTERIOSUS AND PULMONARY HYPERTENSION WHO DIED POSTOPERATIVELY

CASE	AGE (YEARS)	PRESSURE (MM. HG)		SHUNTS		TPR* (DYNES SEC. CM.⁻⁵)
		PULMONARY ARTERY	RADIAL ARTERY	L-TO-R PER CENT PULMONARY FLOW	R-TO-L PER CENT SYSTEMIC FLOW	
21	7½	106/76	100/69	<5	40	1660
26	49	143/78	140/69	25	33	1830
29	24	104/62	127/68	21	30	2140

*Total pulmonary arterial resistance.

The group of patients in whom a right to left shunt has been demonstrated poses a more difficult problem. Because it may provide some insight into the possible selection of patients with right to left shunts for operation, data on 3 of 4 such patients who died after operation and on whom sufficient data are available are analyzed in some detail in Table V. In none of the 4 patients was there any obvious cause of death, and post mortem examination, done in 3, failed to add further information. The calculated shunts represent bidirectional shunt components of pulmonary and systemic arterial blood flow and thus differ from the *net* left to right shunts in Table II, which represent the percentage by which pulmonary blood flow exceeded systemic blood flow. In all 3 of these patients, the right to left shunt exceeded the left-to-right shunt. With the possible exception of 1 case (Case 7), which has been reported in detail elsewhere,[39] there are no late survivors after operation in this group of patients in whom a predominantly right to left shunt was demonstrated. The patient in Case 7 also had a coarctation of the aorta, and for this reason may present a somewhat different hemodynamic problem.

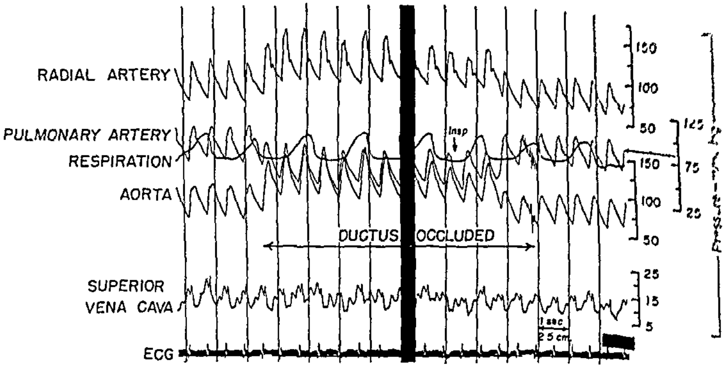

Fig. 4.—Sections of photographic record obtained during operation on a patient with patent ductus arteriosus and pulmonary hypertension (Case 1). Note that during temporary occlusion of the ductus arteriosus there was an immediate fall in pulmonary arterial pressure and a rise in aortic and radial arterial pressures. A few seconds later when the ductus was opened these pressures returned approximately to their previous levels.

An undoubtedly related factor, and one which aids the surgeon in making the final decision regarding closure of the ductus is the hemodynamic response to occlusion of the ductus at the time of operation. The response exhibited to this maneuver in 7 cases is summarized graphically in Fig. 2. Values for pulmonary arterial and systemic arterial pressures before and after closure of the ductus are recorded. A fall in pulmonary arterial pressure and a rise in systemic pressure occurred in all but 2 patients. One patient (Case 21) showed a pronounced rise in pulmonary arterial pressure and a fall in systemic pressure when the ductus was occluded. He survived operation but died suddenly six months later. This case has been reported in detail elsewhere.[30] In Case 7 the patient showed a similar but less marked response to ductal occlusion. This patient has been referred to already as having an associated coarctation.

The hemodynamic response to ductal occlusion, characteristic of the 5 patients with patent ductus arteriosus without associated anomalies who were studied on the operating table and who survived operation and did well postoperatively, consisted of a fall in pulmonary arterial pressure and a rise in systemic pressure. Tracings of the hemodynamic response to this maneuver exhibited in Case 1 on the operating table are reproduced in Fig. 4.

These observations indicate that if it can be demonstrated preoperatively that the shunt is exclusively right to left or that the right-to-left shunt exceeds the left-to-right shunt, surgical closure of the abnormal communication is very hazardous. It is at present our opinion that closure should not be done under these circumstances. Similarly, if, on the operating table, occlusion of the ductus is accompanied by a rise in pulmonary arterial pressure and a fall in systemic pressure, operative closure of the ductus is hazardous and may be unwise. Patients with this disease who have a predominantly left-to-right shunt and who respond to ductal occlusion by a fall in pulmonary arterial pressure and a rise in systemic pressure may be expected to be benefited by operation.

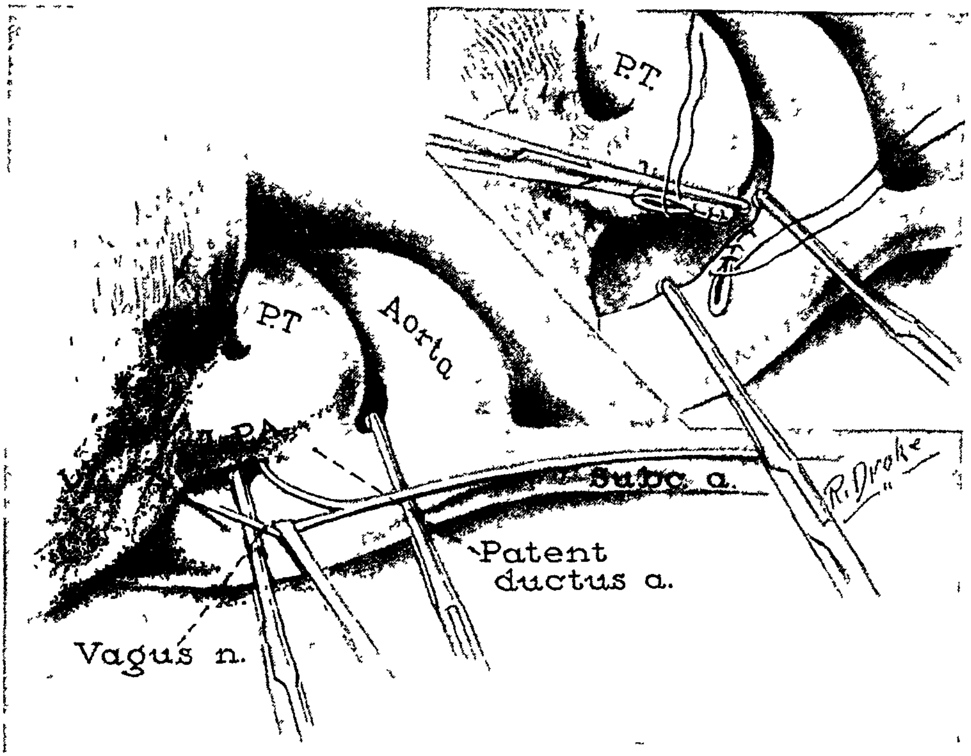

Fig. 5—Technique of division of patent ductus involving cross clamping of the aorta with patient under hypothermia

Operative and Postoperative Management.—Patients with patent ductus arteriosus and pulmonary hypertension usually are found at operation to have an unusually short and extremely wide ductus. Ligation of such a ductus is unsatisfactory, as demonstrated by the fact that of 7 patients treated by this method, recanalization of the ductus was demonstrated in 3. Division of the ductus is the only method of closure for which success can be assured.

Because of the presence of pulmonary hypertension, the pulmonary artery of these patients is large, thin walled, and friable. Alarming hemorrhage can occur with minimal trauma to this vessel. Its wall tolerates the application of clamps poorly. For this reason, every effort should be made to avoid placing clamps in such a manner as to include a portion of the pulmonary artery in their jaws.

Because of our experience with two instances of fatal hemorrhage from the pulmonary artery, the technique of division of the ductus was altered in a recent case. The patient was operated on under hypothermia to 28° C. The aorta was cross clamped above and below the ductus (Fig. 5). The ductus was then clamped at its aortic end, leaving the pulmonary artery end of the ductus and the pulmonary artery undisturbed. The ductus was divided, the cut end of the ductus closed with interrupted 5-0 silk sutures, and the opening in the aorta closed in a similar manner. A similar technique has been described and used by Crafoord and Nylin[51] and Jones[52] in certain cases of patent ductus arteriosus. Hypothermia was not used by these authors. The lengthened period of aortic cross clamping that it will allow adds greatly, we feel, to the ease and safety of the operation.

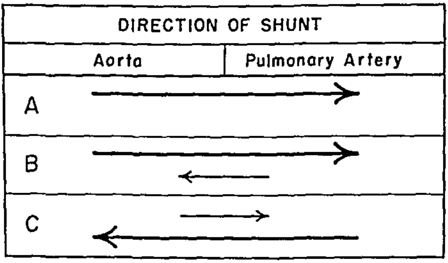

Fig. 6.—Classification of patients with aortic pulmonary communication and pulmonary hypertension.

It has been amply demonstrated that pulmonary vascular resistance is reduced by the inhalation of high concentrations of oxygen.[53] For these reasons it has been our practice to keep all patients who have had operative closure of a patent ductus arteriosus with pulmonary hypertension in an atmosphere of high oxygen concentration for a week to ten days following operation.

CLASSIFICATION

This analysis of 30 cases of aortic pulmonary communication with pulmonary hypertension treated surgically has led to the classification shown in Fig. 6. Separation of patients into the three different categories has certain clinical significance since, at the present time, we believe that all patients in Groups A and *B* should be advised to undergo closure of the ductus. The risks of operation for patients in Group *C* are extremely high, and closure of the ductus in these individuals is probably unwise.

SUMMARY AND CONCLUSIONS

The reported cases of patent ductus arteriosus with pulmonary hypertension have been reviewed. Operative closure of the ductus was attended by a mortality rate of 18 per cent. The mortality rate among patients with a right-to-left shunt was 56 per cent.

In 30 cases of aortic-pulmonary communication with pulmonary hypertension treated surgically at the Mayo Clinic, there were 5 hospital deaths, giving a mortality rate of 17 per cent. All the deaths occurred in patients in whom a right-to-left shunt by way of the ductus had been demonstrated.

A satisfactory drop in pulmonary arterial pressure was noted in those patients who underwent successful closure of the abnormal communication and who were recatheterized in the postoperative period. This satisfactory response included 3 patients in whom a right-to-left shunt was demonstrated preoperatively.

On the basis of the experience gained in the handling of these patients, certain conclusions are justified. Surgical closure of the ductus is hazardous in patients in whom there is demonstrated preoperatively a right-to-left shunt that exceeds in magnitude the left-to-right shunt. Surgical closure of the ductus is hazardous likewise if a rise in pulmonary arterial pressure and a fall in systemic pressure occur in response to ductal occlusion at the time of operation.

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DISCUSSION

DR. FRANK GERBODE, San Francisco, Calif.—I should like to congratulate Dr. Ellis on this presentation and on the excellent manner in which this material was put together. We are in complete agreement with his conclusions; I believe we had reached them, however, before we got into as much operative difficulty as he did in some of his cases. We have encountered at least 20 instances of pulmonary hypertension associated with ductus in approximately 200 patients with patent ductus who were operated upon at Stanford University Hospitals. There were 5 cases among the 20 with what we call a balanced ductus, in which the pressures on the systemic and pulmonary circulations were about the same. In these cases sometimes the shunt was right to left, and in others left to right. One patient had a definite reversed ductus. We have had two cases of reversed ductus in association with coarctation, both of which have been cured by excision and aortic suture. We operated upon these 20 patients without mortality and I would like to show some slides to illustrate the types of cases involved.

(slide) This represents the type in 14 instances in which there was high pulmonary artery pressure, but in which the pressure fell when the ductus was closed. In this instance probably the pulmonary hypertension was related mostly to flow; that is, the amount of blood passing from the systemic to the pulmonary circulation. These patients had excellent recoveries. In quite a few instances lung biopsies showed no changes in the pulmonary vessels, in others definite thickening was found. This is an illustration of the balanced ductus in which we believe that sometimes the flow is from right to left, sometimes from left to right. This boy, as with these other patients, had only a *systolic murmur*. There is a systolic murmur in these patients because the flow usually is not continuous during diastole. They do frequently have a Graham Steell murmur. At the moment the aortic saturation was taken in this instance it was 87 per cent We divided this ductus, as we did the other five, and no difficulties were encountered However, this patient and several others have continued to have a Graham Steell murmur and a loud P , and I believe that perhaps in the future we may see some of these patients coming back to us with so called primary pulmonary hypertension

(slide) Here is another type of balanced ductus in an adult, again a situation in which the pressures on the two sides were the same This woman has gone on to almost complete recovery despite the fact that she had advanced pulmonary arteriosclerosis

(slide) Here is a reversed ductus in a girl who has had considerable cardiac difficulty As you can see, in the operating room with the ductus exposed and shut temporarily, the pulmonary artery pressure rose considerably In fact, the pulmonary artery got extremely tense and we feared it might rupture even while it was temporarily occluded We have always made a practice of observing the change in pulmonary artery pressure with the ductus temporarily occluded in instances of associated pulmonary hypertension This is essential to determine whether or not it is safe to close the ductus If the pressure falls it is safe to go ahead If it rises above that in the aorta it is unwise to close the ductus

(slide) This shows the clinical findings in these patients, clubbing and cyanosis of the toes, and normal appearing fingers This may or may not be present depending upon the permanence and the degree of reversal of flow

DR F MASON SONES, Jr , Cleveland, Ohio—Since 1950, we have encountered 30 patients with severe pulmonary hypertension due to the presence of patent ductus arteriosus None of these patients has shown the continuous murmur clinically associated with this lesion, because of the consistent presence of a diastolic pressure in the pulmonary artery equivalent to, or within, 10 mm of the diastolic pressure in the systemic circuit Eighteen of them were infants less than 2 years of age, 12 were adults

(slide) This is the typical x ray appearance of a very large patent ductus arteriosus in an infant It is clinically impossible to distinguish this lesion from a large intraventricular septal defect

(slide) We have routinely been able to catheterize such ducti As a matter of fact, at the time of heart catheterization it is invariably easier to get into the ductus and demonstrate it anatomically than it is to catheterize the left pumonary artery This is a common diagnostic problem in the infantile group Systolic pressure in the pulmonary artery is significantly lower than that in the aorta but the diastolic pressure gradient is quite small, hence, no continuous murmur In this age group there is invariably a pure and rather large left to right shunt In the adult group much has been said about shunt reversal We would agree entirely with Dr Ellis that the crux of the whole matter depends upon whether or not there is a predominant left to right shunt, or a predominant right to left shunt In all cases in which the shunt is predominantly left to right, even though there may be significant arterial oxygen unsaturation in the aorta below the ductus, we think it is possible to close the ductus, and that no physiologic contraindication to operation exists

(slide) From the anatomic standpoint, however, the surgeon very frequently encounters a situation much like this An extremely short ductus with dilated, sclerotic vessels under extremely high pressure, so that the usual anterior approach with direct attack on the ductus is technically quite hazardous

The simple expedient of radial arteriotomy has been utilized with great success in lowering both systemic and pulmonary artery blood pressure for periods of from thirty to sixty minutes without the development of any arrhythmia, any cardiac complications, and without the necessity for clamping the aorta This is accomplished in adults by the removal of between 500 and 750 c c of whole blood from the artery The process is quickly reversible at any moment you wish to do it The sclerotic vessels actually become smaller, and soften up so that they may be easily and safely mobilized Of course, a posterolateral approach is used with complete mobilization of the aorta above and below the ductus before any direct attempt to immobilize the ductus is made It has been possible by the use of this technique to divide eleven ducti in patients with systemic pulmonary hypertension without immediate or late fatality

DR. ELTON WATKINS, Jr., Boston, Mass.—I would like to congratulate Dr. Ellis, Dr. Kirklin, and Dr. Wood on their excellent presentation, including the very careful physiologic analysis. I felt much better when I saw a slide showing values for pulmonary resistance, in view of the present confusion in the literature between "pulmonary hypertension" and pulmonary vascular resistance.

Concerning this problem, our experience at the Children's Hospital closely parallels theirs. We have the feeling that, with reverse flow through the ductus, operation is definitely not indicated, and that surgery is hazardous with close balancing of the pulmonary and systemic pressures. There is good likelihood that, with the development of pulmonary vascular obstruction, one disease process has been exchanged for another. In place of a ductus defect producing large shunts and increased pulmonary blood flow, we now have pulmonary vascular disease as the major process, and the value of dividing the ductus in such an instance is going to be determined by long-range observations which demonstrate that the pulmonary vascular obstruction is reversible after the shunt is eliminated. It is difficult to visualize how obliteration of a small shunt of a few cubic centimeters will influence the pulmonary vascular process, but it may occur.

So far as the technique of operation goes, we have had good experience lately (in the rare case with an enormous ductus (using the method described by Dr. Freeman of San Francisco, and by Dr. Conklin, of applying a Potts-Smith-Gibson anastomosis clamp to the side of the aorta at the ductus. We have altered their procedure (in patients without pulmonary vascular disease) by opening the pericardium and applying occluding clamps to the left pulmonary artery, thereby achieving complete diversion of the ductus area without blocking aortic flow. This permits easy division and suture of the ductus. We have not had experience with arteriotomy, nor have we found it necessary to use the short-acting autonomic blocking agents, which should achieve the same result as arteriotomy without any tubes or bottles.

DR. H. BRODIE STEPHENS, San Francisco, Calif.—There are a few points I should like to bring up for discussion that Dr. Ellis has probably covered in the manuscript but could not mention today because of lack of time.

I believe the age of the patient may have a considerable bearing upon whether or not the ductus can be divided. I rather believe that pulmonary hypertension develops early in life when the shunt through the ductus amounts to a large volume flow. The pulmonary hypertension may actually be a protective mechanism to reduce the large volume shunt. Division of the ductus early in life may always be possible, assuming that reversal of flow through the ductus is a rare phenomenon under the age of 3 years.

In addition, the presence of associated cardiac septal defect has a significant bearing on the presence of pulmonary hypertension and I am anxious to know what percentage of Dr. Ellis' group had an associated septal defect (particularly those studied at necropsy).

A word with regard to operative management; I believe it is important immediately to mobilize the aortic arch prior to carrying out any dissection in the region of the ductus when pulmonary hypertension is present. By such procedure one has control of the aortic side of the ductus should arrest occur in this structure during its mobilization. It is my practice to clamp the aorta proximal and distal to the ductus after the manner of Crafoord. The ductus is sutured closed as it is divided; this provides improved exposure of the more posterior aspect of the lumen of the ductus and, in addition, lessens the actual time to accomplish the closure.

DR. HARRIS B. SHUMACKER, Jr., Indianapolis, Ind.—This paper which Dr. Ellis and his colleagues have presented certainly will go a long way toward answering many problems which we have all faced and wondered about. We have treated 47 cases of proved pulmonary hypertension of some degree by division of the patent ductus. If we eliminate 3 cases of aortic pulmonary fistula and 8 cases of patent ductus with ventricular septal defect (and I may say that in that particular group our results have been very bad indeed) there remain 36 cases. Of these, only 5 had systemic pressures in the pulmonary artery Of the 21 with pressures less than systemic, all stood division of the ductus well but 1 died of cardiac arrest during bronchoscopy carried out because of atelectasis some time after operation In those with systemic pressure we had 1 death which I feel quite confident was due to a technical error and should have been prevented One patient was catheterized a little over three and one half years after division of the ductus Before operation this child had a pulmonary artery pressure of approximately 100, three and one half years later it was a little over 30

We have used both Arfonad and hexamethonium bromide to produce hypotension with considerable satisfaction, and I believe that this maneuver is of considerable help, particularly in cases with marked pulmonary hypertension

DR ELLIS (Closing) —I should like to thank the discussers for their remarks, and to compliment Dr Gerbode on having avoided some of the pitfalls into which we fell early in our experience with this disease

Associated cardiovascular defects were present in 8 patients Four of these patients had coarctation of the aorta in association with patency of the ductus arteriosus The condition of these patients may present a somewhat different hemodynamic situation, and they have not been considered in detail here They will be presented later as a separate group In addition, there were 2 patients with ventricular septal defects, and 2 with atrial septal defects

One half of the patients were in the pediatric age group, and we have considered that early operation is advisable for these young patients I do not think, however, that the fact that a patient is an adult means that he will not survive operation and do well In fact, one of the most striking decreases in pulmonary arteriolar resistance to follow an operation occurred in a 42 year old patient

Our experience with hypotension as an aid to surgical intervention has been primarily unintentional By that I mean that hypotension has, on occasion, been brought about as a result of hemorrhage during the operative procedure In those cases I could not see that it helped us particularly

I should like to conclude by re emphasizing the fact that the mere presence of a right to left shunt is not a contraindication to operation It is only by careful measurement of the magnitude of these shunts that a decision regarding the advisability of closure of the ductus can be made When the right to left shunt exceeds the left to right shunt closure of the ductus is hazardous and probably should not be carried out Closure of the ductus in patients who have a predominantly left to right shunt is accompanied by an increased risk, for reasons already mentioned, but if closure can be carried out successfully, it should result in clinical improvement for the patient

PULMONARY ARTERIOVENOUS FISTULAS IN SISTERS

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WITH the ever increasing use of mass x-ray techniques in industrial plants, schools, prisons, and hospitals and with their use on large segments of the population by means of mobile x-ray units, "coin lesions" of the lung are being found more frequently. Among the more rare causes of such lesions, one must consider pulmonary arteriovenous fistulas. These fistulas may be small and asymptomatic, as in the two cases presented, or may be large and markedly disabling, and even cause death from hemorrhage, thromboses, or subacute bacterial endarteritis.

Although Churton,[1] in 1897, reported an autopsy in which seven hemangiomas of the lung were found, and Wilkens,[2] in 1918, reported the case of a 23-year-old woman who died of pulmonary hemorrhage and in whom three pulmonary arteriovenous fistulas were found at the post-mortem examination, it was not until the last decade that the medical profession seemed aware of the frequency and possible symptomatic nature of these lesions. It was further brought to their attention by the report of Rodes,[3] in 1938, of a case in which three congenital pulmonary arteriovenous shunts caused polycythemia, disability, and finally death from hemorrhage. The cause of polycythemia was not realized, however, until the post-mortem examination, when it was linked to the intrapulmonary vascular fistulas. In 1939, Smith and Horton[4] reported the first case in which a diagnosis of arteriovenous fistula of the lung was made clinically on the basis of polycythemia and the chest x-ray findings. In 1942, Hepburn and Dauphinee[5] wrote an article describing a case in which a clinical diagnosis was made and in which a pneumonectomy was carried out by Shenstone, thus representing the first surgical cure of this lesion. During the past ten years, about one hundred cases of pulmonary arteriovenous fistulas have been reported in the world literature and of these nearly forty cases have been treated surgically. Goldman[6] has mentioned three reports of pulmonary arteriovenous fistulas present in siblings, including one of his own. To these reports we should like to add another in which the vascular anomaly was present in sisters, one of whom was treated surgically.

These pulmonary vascular lesions have at various times been called cavernous hemangiomas, congenital arteriovenous varices, multiple pulmonary hemangiomas, pulmonary arteriovenous aneurysms, and arteriovenous fistulas of the lung. The latter designation seems most proper since the communica-

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tion between the artery and vein is usually quite direct with no capillary bed between, and it also implies the frequent symptomatic nature of these anomalies. Pulmonary arteriovenous fistula is being rapidly adopted as the correct terminology by the medical profession.

Both of our cases were diagnosed by means of x-ray planography technique which was done following the detection of the lesions on the routine posteroanterior chest x-rays. In the case operated upon there was a vague rounded density, approximately 3 cm. in diameter, superimposed on the anterior portion of the right second rib. Planograms showed the lesion to be at the 8 to 9 cm. level and a vascular marking was seen extending outward from the hilum to the inferior border of this density. The sister who refused surgery presented a rounded density, approximately 2 cm. in diameter, in the left upper lung field and another similar rounded density of the same size in the right lower lung field. Planography revealed a round density in the left upper lobe at the 7½ to 10 cm. level with an afferent and efferent vessel and another density in the right lower lobe at the 5 to 7 cm. level with several vessels entering this lesion. The vascular markings extending into each of these densities as revealed by planography made certain the diagnosis of some kind of vascular lesion.

Further x-ray evidence probably could have been secured by means of fluoroscopy which would have shown the increased pulsations of the hilar structures on the side of the lesion and the pulsations of the lesions themselves. Kymographic studies might also have shown the pulsations in the lesions. X-rays taken with the patient performing the Muller and Valsalva maneuvers might have shown the change in the size of the lesions as the intrathoracic pressure is increased or decreased. Such additional evidence is said to be present in about 50 per cent of instances. Angiocardiography is a valuable procedure for demonstrating multiple lesions not disclosed by other techniques and should be recommended before surgical treatment is instituted in order to prevent overlooking small lesions which would enlarge later as did happen in at least two reported cases.[7]

CASE REPORT

History.—A 53 year old widow entered Episcopal Hospital on Feb. 11, 1954, complaining of sharp right upper quadrant pain which radiated around the costal margin to the back, accompanied by nausea and vomiting of two days' duration. The first such episode had occurred 6 weeks previously, and there were several interval attacks. She had no fever or jaundice. She had had an intolerance to fried and fatty foods for several years.

The patient stated that she had had moderate dyspnea on exertion for the past five years. She was obliged to give up some of her housework, being unable to climb more than one flight of stairs without resting. She had had no ankle edema, palpitations, liver tenderness, or nocturnal dyspnea. She had had almost daily nosebleeds all of her life, but did not consider this serious in so far as her sister, mother, and many aunts, uncles, and cousins also had had such a history of epistaxis throughout their lives. She had had one operation five years previously for a pilonidal cyst.

Physical Examination.—The patient was a slightly obese, middle-aged white woman in no apparent discomfort. Her temperature was 99° F. orally. The blood pressure was

Fig. 1

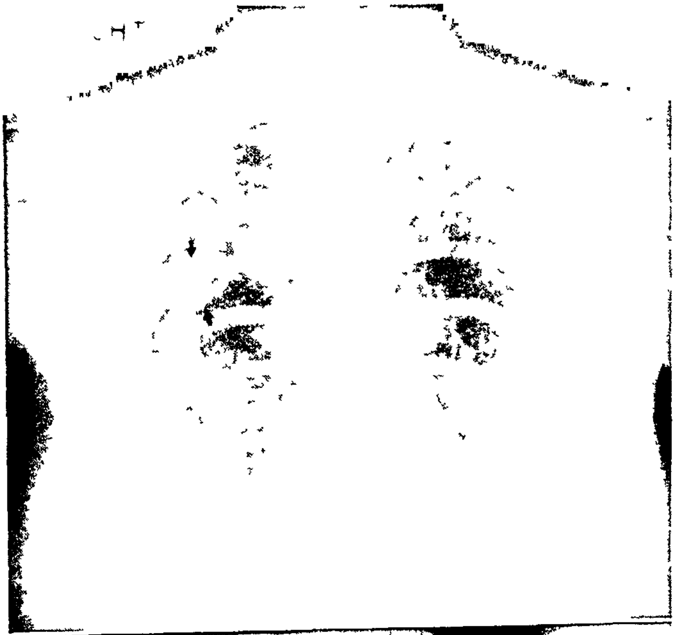


Fig. 2

Fig. 1.—Admission chest x-ray (C. S.) showing rounded density in the right upper lung field.

Fig. 2.—Planogram (C. S.) demonstrating the vessel entering the previously noted density.

100/65, the pulse was 80 per minute, and the respirations were 20 per minute The lips and ears revealed a mild cyanosis There was some ulceration and crust formation over the anterior nasal septum bilaterally The neck was normal The heart sounds were of good quality with a regular rhythm No murmurs were present The chest expanded equally bilaterally and both lungs were clear to auscultation and percussion No rales or bruit were heard The breasts were normal The abdomen was obese and soft There was tenderness to deep palpation in the right upper quadrant The liver edge was one finger breadth below the costal margin No masses were felt The pelvic and rectal examinations were negative The extremities demonstrated no telangiectases or varicosities, but there was moderate clubbing of the fingers and the nail beds were slightly cyanotic The admission diagnoses were cholecystitis with cholelithiasis and possible congenital vascular defect

Laboratory Examinations—On admission, the red cell count was 4 7 million, the hemoglobin was 15 3 Gm, the hematocrit was 47, and the white cell count was 6,250 with 69 per cent polymorphonuclear cells of which 63 per cent were segmented, 27 per cent lymphocytes, and 4 per cent monocytes The prothrombin time was 100 per cent of normal The bleeding time was 1 minute and 30 seconds The coagulation time was 4 minutes and 25 seconds The platelet count was 171,600 The urinalysis was normal The Kline and Kahn tests were negative The blood sugar was 91 mg per cent and the blood urea nitrogen was 15 mg per cent The total protein was 6 1 Gm per cent with an albumin of 3 8 Gm per cent and a globulin of 2 3 Gm per cent The blood calcium was 10 2 mg per cent

A white cell count repeated on Feb 26, 1954, or two days before cholecystectomy, was 14,100 with 84 per cent polymorphonuclear cells of which only 54 per cent were segmented The hemoglobin at this time was 15 7 Gm and the red cell count was 5 09 million *Arterial oxygen saturation was not done*

X-ray Examinations—The admission chest x-ray showed a rounded vague radiopacity 3 cm in diameter in the right hemithorax superimposed on the second anterior rib (Fig 1) Planography was advised to determine the nature of this opacity Planography revealed a density in the right upper lobe at the 8 to 9 cm level with a vascular marking extending from its inferior border to the hilum This suggested the possibility of an arteriovenous fistula (Fig 2) A flat x-ray film of the abdomen revealed vague densities in the right upper quadrant possibly representing calculi and some enlargement of the liver Oral cholecystography was carried out and nonvisualization of the gall bladder resulted No radiopaque calculi were seen

Hospital Course—The patient had been admitted to the medical service and was studied there for gall bladder disease and possible vascular disease The capillary fragility test revealed 40 petechiae in a 1 inch square when the blood pressure cuff was kept above diastolic pressure for 20 minutes A gastrointestinal series to rule out hiatal hernia was negative An electrocardiogram gave no evidence of myocardial infarction After consultation with the Thoracic Surgery Service regarding the pulmonary lesion demonstrated by x-ray, it was decided to proceed with gall bladder surgery initially The patient was then transferred to the General Surgical Service where a cholecystectomy and exploration of the dilated common bile duct were carried out on Feb 17, 1954 The pathological report on the removed specimen was cholecystitis with cholelithiasis The patient had an uneventful postoperative course and was transferred to the Thoracic Surgery Service on March 11, 1954 Exploratory thoracotomy was performed on March 16, 1954

At operation a rounded 2 cm bluish mass was seen projecting from the lateral surface of the lateral segment of the right middle lobe Blood could be seen swirling within this bluish mass and a mild thrill could be felt over its surface This blood flow ceased and the tumor collapsed when the artery to the lateral segment of the middle lobe was occluded The arteries and veins entering the right middle lobe were about three times normal size and there were several large tributary veins entering at the hilum of the middle lobe which made dissection tedious. It was believed that there must also be an arteriovenous fistula in the medial segment of the middle lobe since the arteries and veins entering this segment were also markedly enlarged and a large vein coursed over the inferior surface of this segment. Attention was then directed to the right upper lobe since the opacity seen by x-ray seemed to lie within the anterior segment of this lobe. The vessels entering this segment were two or three times normal size while the vessels to the other segments were of normal size. Pressure over the anterior segment artery made the veins collapse and so it was assumed that an arteriovenous fistula lay within the segment. The artery to this segment of the upper lobe was merely ligated without removing the segment so that pulmonary tissue might be spared. The right middle lobe was removed by the individual ligation technique.

Fig. 3.

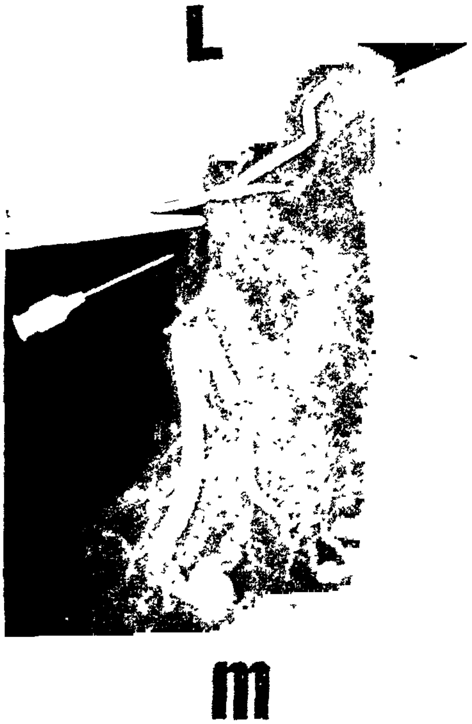
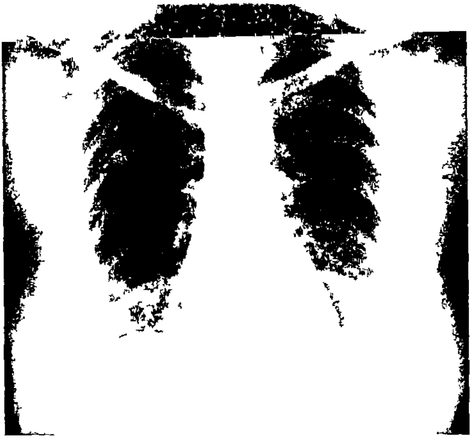


Fig. 3.—Right middle lobe injected with Lipiodol. Hemostat is in the bronchus, catheter in the artery, and needle in the vein. The longer needle indicates the subserosal vascular sac from which some dye has escaped. Three arteriovenous fistulas are seen in this injected specimen (C. S.).

The microscopic slides of the arteries and veins of the removed right middle lobe showed nothing unusual. The bluish mass on the lateral segment was a thin-walled venous sac. The specimen was injected with Lipiodol and x-rays were taken (Fig. 3). The injected specimen showed two direct arteriovenous shunts in the medial segment of the right middle lobe and one in the lateral segment which entered the superficial thin-walled vascular sac.

The patient had an uneventful postoperative course except for a low-grade fever. The chest drainage tubes were removed on the fourth postoperative day. Fluid accumulation necessitated a thoracentesis on one occasion. The sutures were removed on the seventh postoperative day and the patient was discharged to her home on the tenth postoperative day. The discharge chest x-ray showed no remaining radiopacity in the lung fields and the mild cyanosis seen on admission had disappeared.

Fig 4



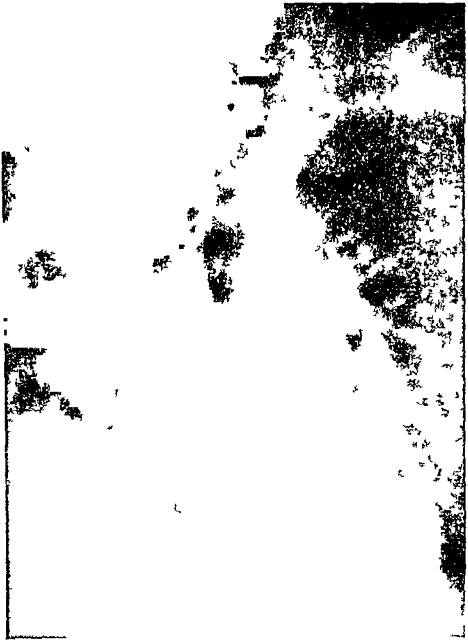


Fig 5

Fig 4 —A follow up chest x ray (C S) two and one half months after the first operation reveals a density in the same location as previously noted in the right upper lung field

Fig 5 —Pulmonary angiogram (C S) revealed the density to be a remaining arteriovenous fistula

When seen on a follow-up examination on May 7, 1954, an opacity was again noted in the right upper lobe by x-ray (Fig. 4). In order to determine if this was the arteriovenous fistula which had been ligated but not removed at the first operation, a pulmonary angiogram was performed on July 7, 1954. This showed a large arteriovenous fistula to be present still in the anterior segment of the right upper lobe (Fig. 5). This was presumed to be the same fistula in which the vessels were ligated at the first operation.

Second Hospital Admission.—The patient re-entered the hospital for excision of the remaining arteriovenous fistula, and on July 17, 1954, the anterior segment of the right upper lobe was removed. This segment was connected by many vascular channels to the parietal pleura and the adjacent segments, and a thrill could be felt over the segment. The removed segment was injected with Lipiodol and an x-ray was taken. It showed a dilated vascular sac which almost completely filled the segment (Fig. 6). The five veins which drained the sac are not seen, since in order to fill the sac with the radiopaque material, they had to be ligated.

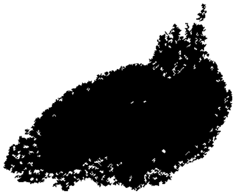


Fig. 6.

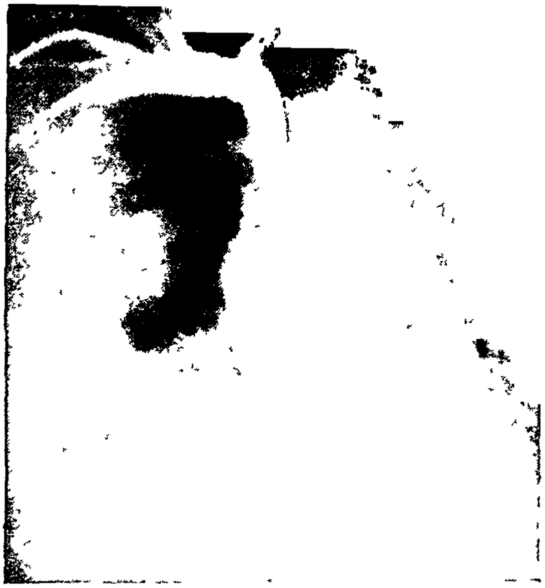


Fig. 7.

Fig. 6.—Lipiodol injection of the anterior of the anterior segment of the right upper lobe reveals it to be almost completely replaced by the sac of a large arteriovenous fistula (C. S.).

Fig. 7.—A postoperative pulmonary angiogram (C. S.) reveals no remaining arteriovenous fistulas. The haziness over the lower right lung field is due to pleural thickening in that area.

A repeat pulmonary angiogram was done ten days after the second operation. This did not show any remaining arteriovenous fistulas (Fig. 7). Pleural thickening gives a slight haze over the lateral aspect of the right lung field.

The patient is now doing her own housework and exhibits no cyanosis. Her hemoglobin and red cell count are within normal limits.

Knowing the hereditary nature of arteriovenous fistulas of the lung and having obtained a history of epistaxis in many other members of the family, it was decided to obtain a chest x-ray of as many members of the family as possible who lived in this area. A sister, a brother, and two cousins were x-rayed. The chest x-ray of the brother was negative and he had no clubbing of the fingers, no telangiectases of the skin or mucous membranes, and no history of epistaxis. One female cousin had telangiectases over the abdomen, but no history of epistaxis and no clubbing of the fingers Her chest x ray was negative Another male cousin gave a history of frequent episodes of epistaxis since childhood but his chest x ray was negative One sister of the patient had a history of frequent nosebleeds since earliest childhood She had moderate clubbing of the fingers, mild cyanosis, and telangiectases of the lips Her chest film revealed a rounded density in the upper left and lower right lung fields, each about 2 cm in diameter (Fig 8) Planograms showed vessels entering each of these lesions (Fig 9) She denied dyspnea on exertion but actually seldom exerted herself, probably because of her known limitations She was advised to have surgical excision of these lesions because of the dangers of their enlargement with possible hemorrhage, thromboses, or subacute bacterial endarteritis She has refused operative intervention

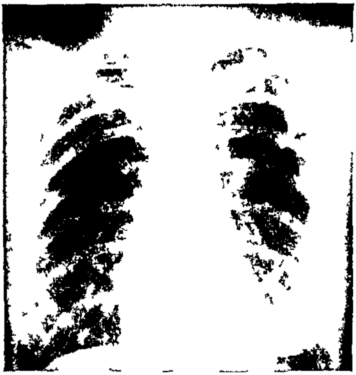


Fig 8—The chest x ray of the patient s sister (M S) reveals densities in the left upper lung field and right lower lung field

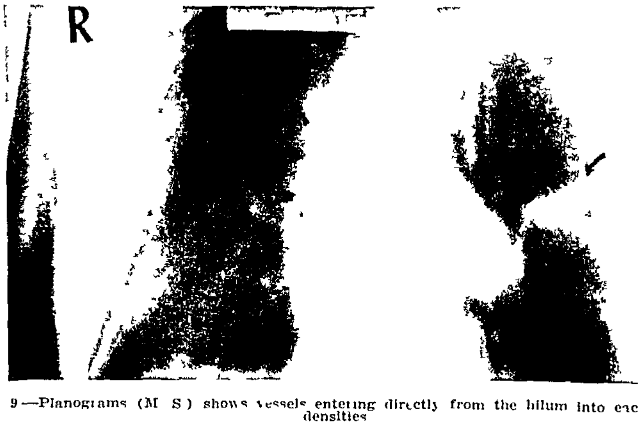
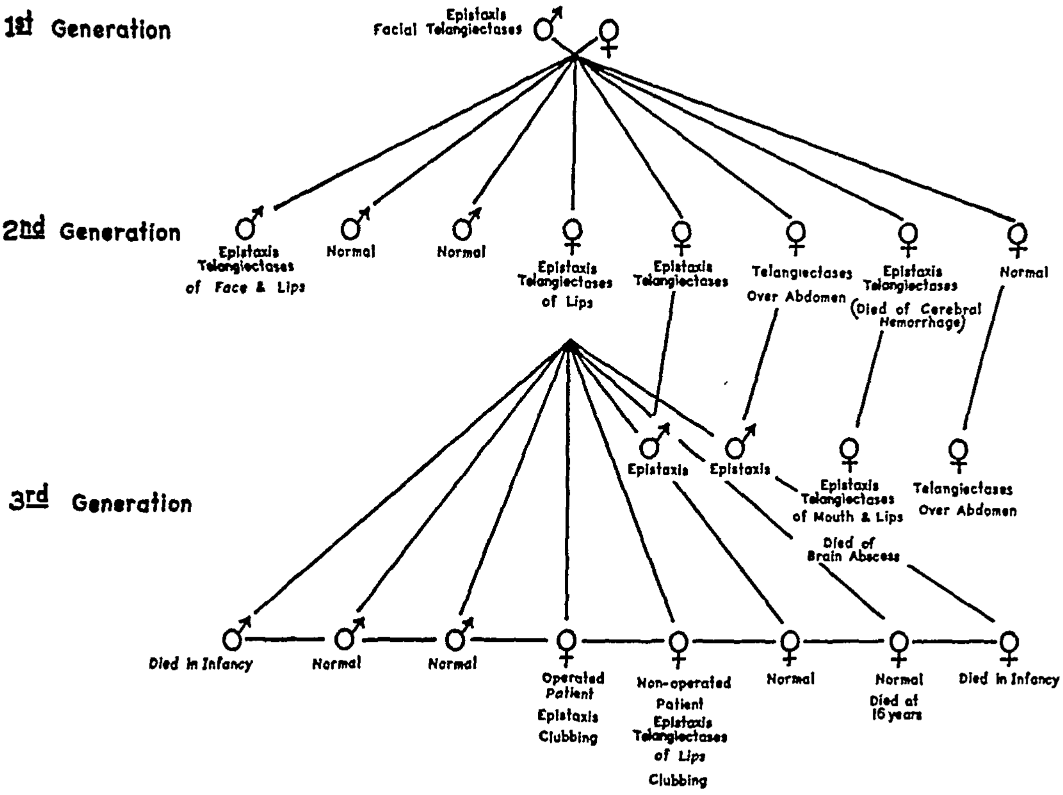


Fig 9—Planograms (M S) shows vessels entering directly from the hilum into each of the densities

COMMENT

Pulmonary arteriovenous fistula is, in nearly all instances, a manifestation of hereditary hemorrhagic telangiectasia. Hereditary hemorrhagic telangiectasia, or Rendu-Osler-Weber disease, is an interesting congenital vascular anomaly which is transmitted as a simple dominant. It is not sex-linked, although some have felt that it is more common in females than males. The manner in which it has passed through three generations in this family is shown in Fig. 10. The telangiectases are usually seen on the skin of the face and neck and on the buccal and nasopharyngeal mucous membranes. Epistaxis is the most common symptom but often does not appear before the age of puberty. The lesion is fundamentally a congenital defect of development of the terminal loops of the capillaries with hemorrhage due to the fragility of the small vessels. The coagulation time, bleeding time, platelet count, and fragility tests are all normal. There may be telangiectases in any of the abdominal viscera, the lungs, the mucous membranes of the gastrointestinal, respiratory, or genitourinary tracts, and even the brain or spinal cord. Hemorrhage from these telangiectases may give epistaxis, hematemesis, hemoptysis, melena, hematuria, or meningeal irritation. Hemorrhage into some vital organ or exsanguinating


1st Generation
Epistaxis
Facial Telangiectases
♂
♀
2nd Generation
♂ Epistaxis Telangiectases of Face & Lips
♂ Normal
♂ Normal
♀ Epistaxis Telangiectases of Lips
♀ Epistaxis Telangiectases
♀ Telangiectases Over Abdomen
♀ Epistaxis Telangiectases (Died of Cerebral Hemorrhage)
♀ Normal
3rd Generation
♂ Epistaxis
♂ Epistaxis
♀ Epistaxis Telangiectases of Mouth & Lips
♀ Telangiectases Over Abdomen
Died of Brain Abscess
♂ Died in Infancy
♂ Normal
♂ Normal
♀ Operated Patient Epistaxis Clubbing
♀ Non-operated Patient Epistaxis Telangiectases of Lips Clubbing
♀ Normal
♀ Normal Died at 16 years
♀ Died in Infancy

HEREDITARY HEMORRHAGIC TELANGIECTASIA IN THREE GENERATIONS OF THE FAMILY OF THE PRESENTED CASES

Fig. 10.—Genealogy of the patient's family.

hemorrhage may be the cause of the patient's death Sudden death in infancy in two of the patient's siblings could have been due to internal hemorrhage, although there was no outward manifestation of bleeding and the deaths were not observed by a physician

Wells[8] has written of the possible overlap of hereditary hemorrhagic telangiectasia, hereditary familial vascular purpura, and pseudohemophilia in a very small proportion of cases We believe that both hereditary hemorrhagic telangiectasia and hereditary familial vascular purpura are present in our two patients since they both have visible telangiectases as evidence of the former and a positive tourniquet test and increased capillary fragility as evidence of the latter Pseudohemophilia, which causes an increased bleeding time and deficient contractility of the clot, is not present

The altered physiology caused by a pulmonary arteriovenous fistula differs in some fundamental aspects from such a fistula in the systemic circulation Maier, Himmelstein and co-workers[9] have brought these differences to the attention of the medical profession and their clinical investigations have been corroborated by the studies of Takaro, Essex, and Burchell[10] who created artificial direct fistulas between the pulmonary arteries and veins in dogs In both peripheral arteriovenous fistulas and pulmonary arteriovenous fistulas the total blood volume is increased, but in the pulmonary arteriovenous fistulas the total plasma volume is not increased as in the systemic form There is thus a much greater increase in the red cell mass in the pulmonary type without a concomitant increase in plasma volume This results in a great increase in the blood viscosity and there is a greater tendency toward the formation of thromboses in the pulmonary type The hematocrit, therefore, is also greatly increased in the large symptomatic pulmonary arteriovenous fistulas, whereas it is normal in systemic arteriovenous fistulas Oxygen saturation is normal in the systemic form while it is decreased to about 70 per cent in the pulmonary form, thus accounting for the cyanosis with even small pulmonary shunts The absence of cyanosis in the face of a vascular shunt seen in the lung fields by x-ray would probably mean a shunt between the bronchial arterial system and the pulmonary veins since the bronchial arteries have no connection with the pulmonary arteries but both drain into the pulmonary veins Cardiac output is normal in the pulmonary shunts, but it is increased in the systemic type The cardiac enlargement seen in the systemic fistulas does not occur with pulmonary fistulas Thus, one may summarize the physiological changes of a pulmonary arteriovenous fistula by stating that it increases the red cell mass only and has little or no effect on cardiac output and cardiac dynamics, while the peripheral arteriovenous fistula increases all the elements of the blood volume proportionately and causes an increased cardiac output and dilatation of the heart

The polycythemia is marked in large pulmonary fistulas and leads to increased viscosity of the blood and thromboses The cerebral anoxia and cerebral thromboses cause symptoms of headache, vertigo, convulsions, and even paresis An aunt of the patient had a history of epistaxis almost daily and telangiectases of the face, neck, nose, and mouth. She developed headaches and dizziness. At another hospital she was explored for a possible brain tumor and died of intracerebral hemorrhage following the evacuation of a cerebral clot. The chest x-ray findings in this case are not reported.

Subacute bacterial endarteritis is another possible complication of pulmonary arteriovenous fistulas. This was present in the case reported by Maier, Himmelstein and associates[9] and resulted in a brain abscess in the cases reported by Wodehouse, and by Stern and Naffziger.[11] A cousin of our patient, the daughter of the patient mentioned previously who had died of cerebral hemorrhage, died of an intracerebral abscess at 22 years of age in another hospital. She had had a history of daily nosebleeds and had telangiectases of the nasal and buccal mucosa. She was admitted, suspected of having a brain tumor but died while studies were being carried out. Autopsy revealed a right frontoparietal abscess.

Some cases of polycythemia vera could be mistaken for a pulmonary arteriovenous fistula. The differential points here would be the following findings in polycythemia vera: an enlarged spleen, the absence of clubbing of the fingers, the elevated white cell count with immature white cells prominent, and an arterial oxygen saturation which is normal or nearly so. There may be vague densities in the lung fields with polycythemia vera but these are not constant as in the case of a pulmonary arteriovenous fistula. The white cell count, maturity of the white cells and spleen are all normal with a pulmonary fistula but the oxygen saturation is about 70 per cent.

It is interesting that, in spite of the four large direct shunts found in our case at operation, she exhibited only mild cyanosis and had hardly any elevation of the hemoglobin, red cell count, or hematocrit. Perhaps her almost daily epistaxis was a factor in the lower than expected red cell mass. The patient stated that as a child her mother kept a pail in the kitchen for the many handkerchiefs used for the episodes of epistaxis by her mother, her sister, and herself. These handkerchiefs were washed out daily in large numbers. Goldman as well as Glenn, Harrison, and Steinberg[12] have described cases in which the hemoglobin and hematocrit were not elevated. The low red cell mass in these cases was attributed to the recurrent epistaxis or other causes of anemia. Both our patient who was operated upon and her sister had mild cyanosis. When the red cell count is normal a shunt of 25 to 30 per cent of the blood volume is necessary in order to give cyanosis. Neither of our patients demonstrated the continuous purring murmur, with accentuation at cardiac systole and with deep inspiration, heard over the periphery of the lung in about 50 per cent of the recorded cases.

Another possible source of confusion in the differential diagnosis is congenital heart disease. Any heart lesion producing cyanosis, clubbing, and polycythemia must be serious and would give cardiac murmurs and changes in the cardiac outline by x-ray. In pulmonary arteriovenous fistulas the heart is normal by auscultation and x-ray. The arm-to-tongue circulation time is actually decreased because of the pulmonary shunt and one may get peripheral manifestations of the ether injected for the arm to lung test, such as tingling in the face and scalp, because the ether goes through the pulmonary shunt without being vaporized entirely in the lung capillary bed which normally occurs [13]

SUMMARY

The fourth report of pulmonary arteriovenous fistulas appearing in siblings has been given, and another case has been added to the growing number of reported arteriovenous fistulas of the lung which have been treated surgically The sister of the patient operated upon was shown also to have pulmonary arteriovenous fistulas but she has refused operative intervention Both women were members of a family in which Rendu Osler Weber disease was known to be present for at least three generations The physiologic changes caused by pulmonary arteriovenous fistulas and their differential diagnosis have been discussed

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THE TREATMENT OF METASTATIC PULMONARY MALIGNANCY

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SIGNIFICANT advances in all phases of surgical management during the past few decades have gradually allowed the surgeon to re-evaluate a number of conditions previously considered therapeutically unapproachable. Nowhere is this trend more evident than in the field of neoplastic disease, where reduction in operative mortality, together with the development of increasingly adequate methods of dealing with the primary malignancy and its local extension, has led to the accumulation of a steadily growing number of patients in whom the primary lesion has been controlled, but who present metastatic growth elsewhere in the body. It was inevitable that this situation should result in attempts at direct attack, aimed at eradicating these secondary lesions by procedures which, although certainly feasible from a technical standpoint, were approached originally with a certain sense of desperation. The acceptance of these procedures as rational and effective therapy has undoubtedly lent impetus to the consideration of direct attack on metastatic disease of the lung. Formerly considered evidence of a surely fatal prognosis, secondary pulmonary malignancy, in the light of the experience of the past two decades, can be shown to be controllable in a small but significant number of patients.

Seiler,[8] in 1949, in a comprehensive review, presented data on 62 such cases, including 10 of his own, with follow-up periods ranging from one month to eighteen years. During the discussion following this paper, 12 additional cases were contributed by Van Hazel,[8] Waterman,[8] and Lambert,[8] with six more cases being added by the Mayo Group. Further reports were made by Ehrenhaft,[4] and by Hilton and associates,[5] in 1951, and by Mannix,[7] in 1953. A total of 91 cases are therefore available from the literature.

In reviewing our own experience at the Hines Veterans Administration Hospital, a total of 22 patients were found who had been explored for supposed metastatic lesions. Eighteen of these lesions were proved microscopically to be definite metastases (Table I), while the remaining 4 patients were shown to have definite or highly probable primary pulmonary malignancies (Table II). These latter cases will be discussed separately. Thus, there are at present 109 known instances of pulmonary resection for metastatic lesions. Of these patients, 82 have been followed for two years or longer, of whom 63 have died, giving a two-year survival without recurrence of 19 (23.2 per cent).

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TABLE I PULMONARY RESECTION FOR METASTATIC MALIGNANCY (DATE OF COMPILATION, DECEMBER, 1953)

CASE	PRIMARY LESION	OPERATION	TIME INTERVAL (MONTHS)		SURVIVAL (MONTHS) POSTRESECTION		CAUSE OF DEATH
			PRIMARY TO METASTASIS	METASTASIS TO RESECTION	LIVING	DEAD	
1	Adenocarcinoma Rectosigmoid	Lobectomy	23	-0		64	Carcinoma, pancreas
2	Adenocarcinoma Right kidney	Segmental resection	60	4	13		
3	Teratoma Left testicle	Wedge resection	57 66*	1 1	 13		
4	Adenocarcinoma Site unknown	Pneumonectomy	Unknown	9	9		
5	Transitional carcinoma Right kidney	Wedge resection	0	1½		15	Congestive heart failure
6	Adenocarcinoma Rectum	Pneumonectomy	39	1		6	Recurrent tumor
7	Hypernephroma Left kidney	Wedge resection	0	3		5	Recurrent tumor
8	Adenocarcinoma Rectosigmoid	Lingulectomy	30	17		14	Recurrent tumor
9	Chondrosarcoma Left femur	Wedge resection	13	2		7	Recurrent tumor
10	Osteogenic sarcoma Right tibia	Segmental resection	7	5		33	Recurrent tumor
11	Synovioma Right forearm	Lobectomy	5	1		6	Recurrent tumor
12	Rhabdomyosarcoma Left thigh	Lobectomy	14	2		11	Recurrent tumor
13	Rhabdomyosarcoma Right lateral chest	Wedge resection	16	15		18	Recurrent tumor
14	Fibrosarcoma Left pectoral	Wedge resection	12	1		18	Recurrent tumor
15	Chondrosarcoma Left femur	Wedge resection	4	4		9½	Recurrent tumor
16	Chondrosarcoma Right fibula	Wedge resection	13	1½		11	Recurrent tumor
17	Adenocarcinoma Colon	Lobectomy	96	1	1		
18	Epidermoid carcinoma Piriform sinus	Lingulectomy	21	9	4		

*Second metastasis resected

TABLE II. SECOND PRIMARY PULMONARY MALIGNANCIES

CASE	ORIGINAL PRIMARY	INTERVAL BETWEEN PRIMARIES (MONTHS)	SECOND PRIMARY	OPERATION	SURVIVAL (MONTHS)	
					LIVING	DEAD
19	Squamous-cell carcinoma Larynx	15	Right upper lobe	Inoperable	2	
20	Adenocarcinoma *Colon*	34	Left lower lobe	Pneumonectomy		4
21	Epidermoid carcinoma Kidney-pelvis	0	Superior segment left lower lobe	Pneumonectomy		8
22	Adenocarcinoma Stomach	0	Right upper and middle lobes	Bilobectomy		0

It is our purpose in this presentation to emphasize the fact that in patients in whom there is reasonable evidence that a primary malignancy has been controlled, the presence of a discrete *pulmonary lesion is an absolute indication* for thoracotomy. Not only is there a fair chance of controlling the metastatic lesions, but the real possibility that a second primary pulmonary malignancy may coexist is ever present. The first point for consideration in this group is, then, the present status of the primary lesion. Obviously, to the patient with local persistence of disease, or with metastases in other organs, excision of the pulmonary lesion offers no real benefit. Indiscriminate exploration, even in the face of a hopeless prognosis, then becomes senseless mutilation. Unfortunately, *the only criteria presently available for evaluating the operability of these patients are those obvious factors which are immediately apparent on considering* the subject. We can only hope that as further experience is accumulated, more specific factors may appear, allowing a more realistic selection. In spite of this, and especially in view of the significant survival figures shown above, we believe that adherence to the following criteria will prove rewarding.

CRITERIA FOR EXPLORATION

Control of the Primary.—In any such case coming to exploration, it must first of all be established beyond reasonable doubt that the original cancer has been eradicated. Tumors of the extremities, breast, oral cavity, and the like, by their location, lend themselves to easier evaluation. Where any doubt may exist they are readily accessible to biopsy. Alexander and Haight[1] report biopsying the primary site because of induration and ulceration following radiation therapy in a man who had had a fibrosarcoma of the thigh locally excised twenty-one months previously. This patient is known to have survived beyond eleven years following lobectomy. Abdominal cancer on the other hand must be evaluated by less tangible factors. The type and grade of malignancy, together with the findings at the original operation and the type of primary treatment used must all be considered. Ideally, a sufficient time interval should have elapsed between the primary treatment and the appearance of metastases to form some judgment on the basis of the expected course of the tumor in question. Unfortunately metastases are frequently present at the time the primary growth is first diagnosed, or shortly thereafter, and the decision must then, of necessity, be made on less satisfactory evidence.

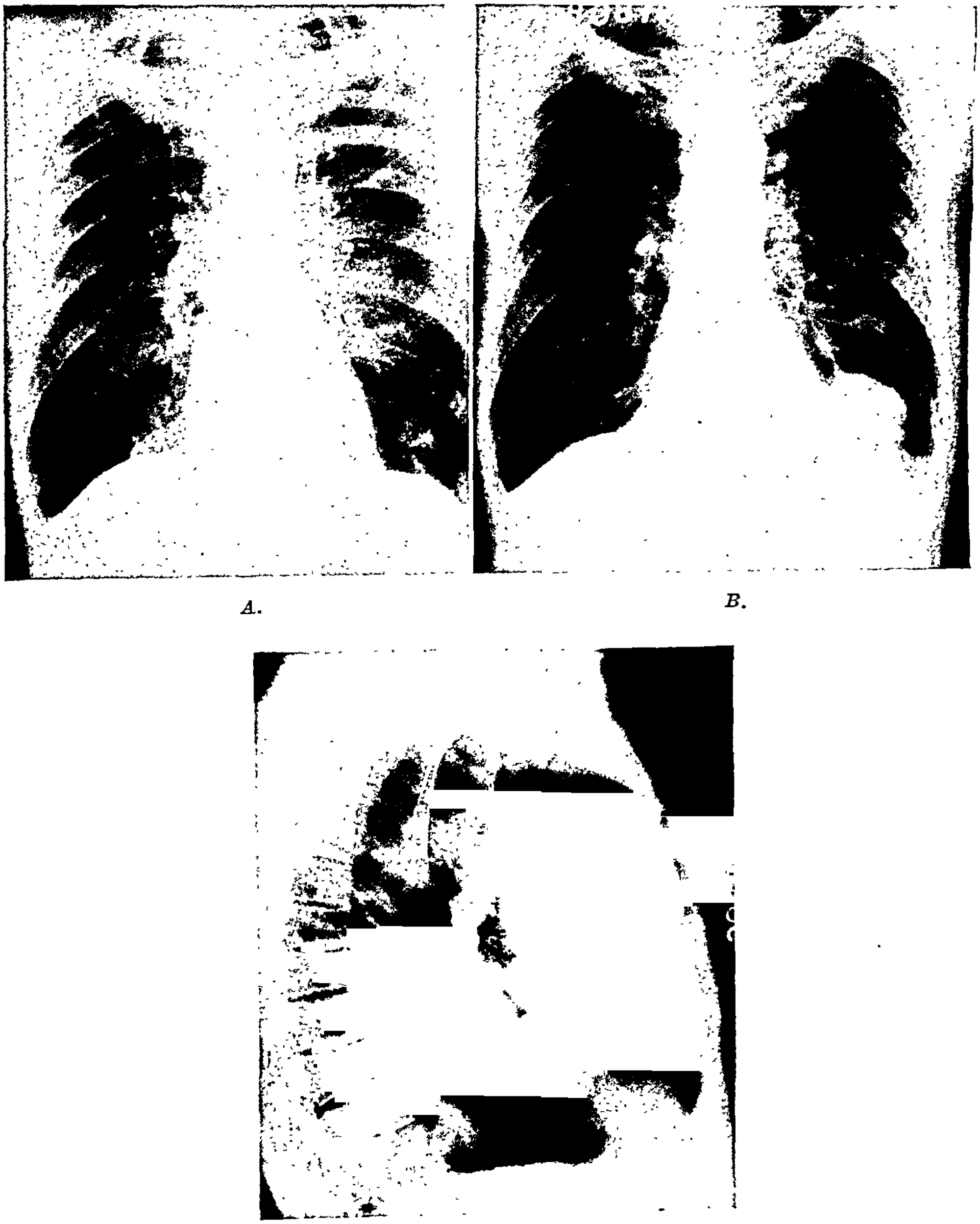
Absence of Other Metastases.—A careful examination should be undertaken to determine the presence of other metastases. This should include especially the regional lymphatics, central nervous system, skeleton, and any other areas for which the primary has a known predilection. While metastases in two different locations materially worsen the prognosis, even this situation may be amenable to surgical intervention in rare instances. Effler and Blades[3] report a case in which a lobectomy for carcinoma, metastatic from the sigmoid, was followed five months later by the removal of an intracranial metastasis. This patient was alive and well one and one half years following the pulmonary resection.

Accessibility to Resection.—The ideal lesion for resection is a single, discrete metastasis confined to one lobe or segment. The presence of more widespread pulmonary involvement does not of itself contraindicate exploration, provided the patient has sufficient pulmonary reserve to withstand more extensive resection. On the other hand, the presence of recurrent laryngeal or phrenic nerve involvement, pleural effusion, or direct extension to the chest wall, usually signifies unresectibility and these should generally be considered as contraindications to exploration. While the uncomfortable possibility that other small undetectable metastases may coexist is ever present, this risk must be accepted where the other factors indicate a good chance of success. In this connection, the use of chest laminography is extremely useful in demonstrating metastases which may not be apparent on conventional chest films.

Type of Primary Tumor.—Analysis of the 109 reported cases reveals that 65 were carcinomas and 38 were sarcomas. In 6 cases, the primary was either unknown or not stated. On the other hand, among the 19 patients who survived two years or longer, there were 8 carcinomas and 11 sarcomas. In view of the normal preponderance of primary carcinoma over sarcoma, these figures indicate that localized, accessible metastases occur more frequently in the sarcomatous group. Also, the two year survival figures showing a greater number of sarcomas despite a smaller original group indicate a more favorable prognosis. However, the small number of cases in this series probably does not allow tumor type to be used as an adequate criterion.

The following case illustrates the ideal situation in which the primary has been well and adequately dealt with, and a solitary pulmonary metastasis, which is surgically accessible, represents the only remnant of disease. Admittedly, the incidence of such cases is low, yet the evidence that a good result is obtainable imposes the obligation to evaluate all possible cases for surgical intervention.

CASE 1.—F. H., a 66 year old man, was first admitted to Hines Hospital on Sept. 14, 1942, with a diagnosis of rectal tumor. Biopsy revealed an adenocarcinoma, and on Sept. 21, 1942, an abdominoperineal resection was performed with an uncomplicated postoperative course. He was readmitted in August, 1944, with complaints of melena and upper abdominal pain. Gastrointestinal x-rays revealed a duodenal ulcer and on medical management there was prompt disappearance of symptoms. There was no evidence of local recurrence of the rectal malignancy; however, a rounded, discrete mass, felt to represent a metastasis, was noted in the left lower lobe (Fig. 1, *A*). At this time, he was transferred to another institution for domiciliary care. He was again readmitted on April 17, 1946, because of intermittent bouts of diarrhea, a complaint which subsided promptly with dietary adjustment. Chest x-ray at that time showed the lesion in the left lower lobe to have increased considerably in size (Fig. 1, *B* and *C*). Since he had then passed three and a half years without evidence of local recurrence, and the pulmonary lesion, if metastatic, was possibly the sole residual of the



A. *B.*

C.

Fig. 1.—Case 1.

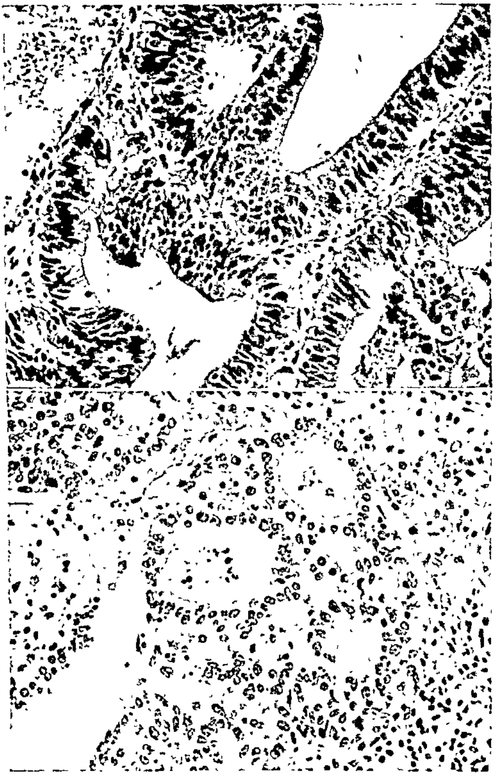
A, Chest x-ray in August, 1944. A discrete lesion is seen in the left lower lobe just above the diaphragm.

B, Chest x-ray, April, 1946. The lesion has progressed.

C, Chest x-ray in lateral view showing the lesion to be in the lower lobe.

disease, it was decided that an attempt at removal should be made. On April 30, 1946, a left lower lobectomy was performed with an entirely uneventful postoperative course. The histologic sections confirmed the diagnosis of metastatic rectal adenocarcinoma (Fig. 1, *D* and *E*). He was then returned to domiciliary care, where he died on August 29, 1951, having survived five and a half years following the removal of the metastatic lesion and nearly nine years (eight years and eleven months) following his primary surgery. Autopsy revealed death to be due to a caseating tuberculous pneumonia and carcinoma of the tail of the pancreas with liver metastases.

D.



E.

Fig. 1 (*Cont'd*).

D, Photomicrograph in primary tumor of the rectum, showing a rather typical adenocarcinoma.

E, Photomicrograph of the pulmonary lesion shows a less well-differentiated tumor but the preservation of the rather typical pattern confirms its metastatic nature.

SECOND PRIMARY LESIONS

Hood and co-workers,[6] in a recent review of a large group of solitary, circumscribed pulmonary masses, showed an incidence of 42 per cent malignancy in noncalcified lesions, and emphasized the importance of exploration in all such cases. One may reason from these statistics and from the increasing incidence of pulmonary carcinoma in recent years, that even in patients with a prior history of malignant disease a circumscribed pulmonary lesion will, in a certain percentage, represent a primary tumor of lung. That the percentage of these second primaries may actually be significantly high became apparent to us in reviewing our material. Our original group of patients explored for supposed pulmonary metastases numbered 22. Of these, only 18 lesions could be

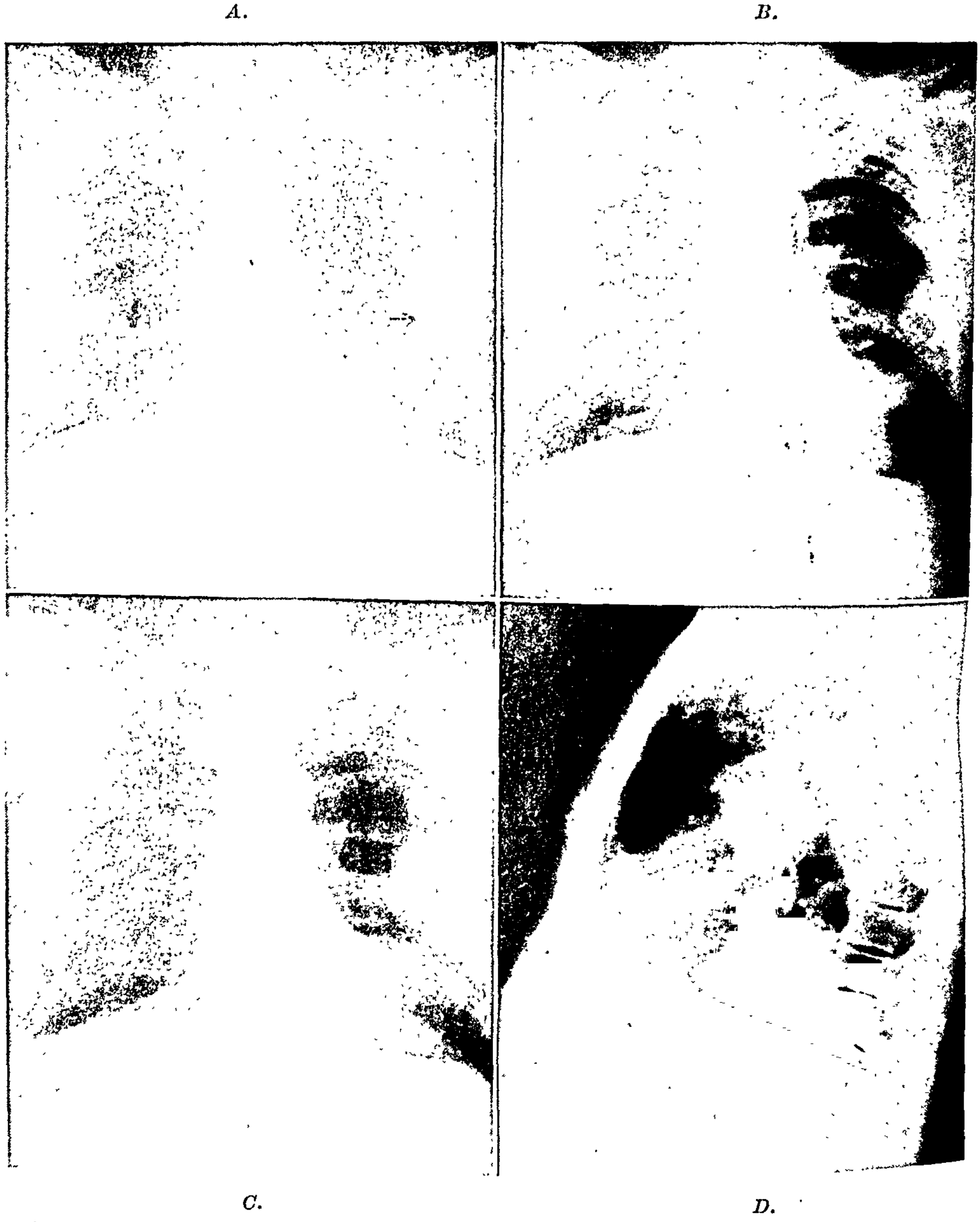


Fig. 2.—Case 2.

A, Chest x-ray of September, 1950, showing a small lesion in the left fourth anterior interspace.

B, Chest x-ray of October, 1951, showing progression of the lesion. It is now very plainly seen.

C, Chest x-ray of June, 1952, showing further progression.

D, Chest x-ray in lateral view of *C*.

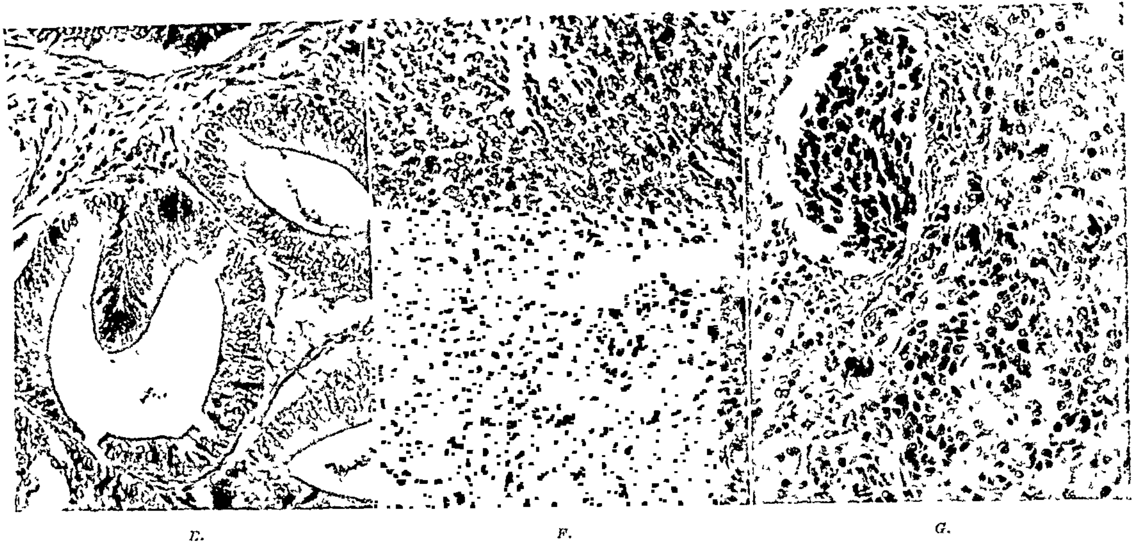


Fig 2 (*Cont'd*)

E, Photomicrograph of the original tumor in the ascending colon, representing a typical colonic adenocarcinoma

F, Photomicrograph of pulmonary tumor, bearing no similarity to the original colonic neoplasm. This is a highly undifferentiated tumor arising in lung

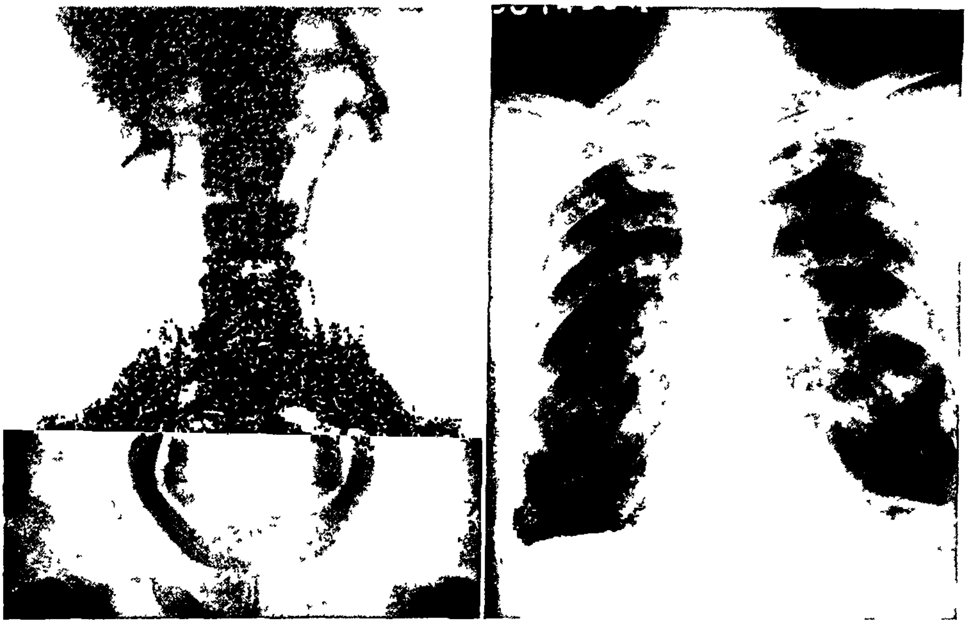
G, Section of liver showing a metastatic lesion identical to the pulmonary tumor.

definitely established as metastatic. Of the remaining 4 cases, 3 are well substantiated as primary pulmonary lesions, while the remaining case, by its clinical course and histologic picture, is far more likely to be primary than secondary. This is an incidence of 18.2 per cent primary lesions in our series (Table III). We are herewith reporting these 4 cases in detail.

TABLE III. ALL PULMONARY LESIONS

TOTAL CASES	METASTATIC LESIONS		SECONDARY PRIMARY LESIONS	
	NUMBER	PER CENT	NUMBER	PER CENT
22	18	81.8	4	18.2

CASE 2.—W. C., a 58-year-old man, was readmitted on June 4, 1952, with complaint of a chronic productive cough of several months' duration. He had been previously hospitalized, in 1948, at which time a right hemicolectomy was performed for adenocarcinoma of the ascending colon (Fig. 2, *E*). He then remained asymptomatic until shortly before his second admission. Chest film of May 20, 1952, revealed the presence of a discrete mass in the periphery of the left lower lung field. Review of past films showed the lesion to have been first visible one year and nine months previously, although unidentified at that time (Fig. 2, *A*, *B*, *C*, and *D*). On June 26, 1952, a left pneumonectomy was carried out, and the histologic sections showed an undifferentiated bronchogenic carcinoma (Fig. 2, *F*). He died on Oct. 18, 1952. An autopsy revealed the cause of death to be massive liver metastases from the bronchogenic carcinoma (Fig. 2, *G*). No evidence of remaining adenocarcinoma could be found.

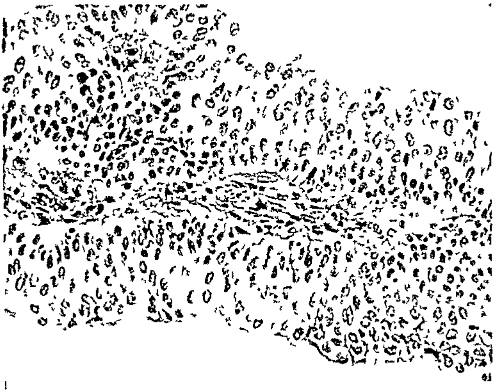


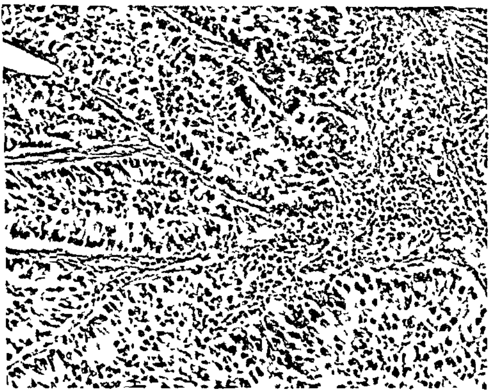
A. *B.*

Fig. 3.—Case 3.
A, Retrograde pyelogram A filling defect is noted in the left renal pelvis
B, Chest x-ray reveals a discrete lesion partially overlying the left fourth anterior rib.

CASE 3.—C. P., a 56-year-old white man, was admitted to the hospital on March 19, 1951, in cardiac decompensation with a history of a chronic cough and progressive dyspnea of two weeks' duration. At the time of admission, he was acutely ill, requiring nasal oxygen and rapid digitalization. He responded rather rapidly, however, and by the end of one week the heart was well compensated. Chest x-ray at that time showed a rounded 3 cm. mass in the superior segment of the left lower lobe (Fig. 3, *B*). Blood chemistry, sputum examination, and bronchoscopy were nonrevealing; however, an intravenous pyelogram demonstrated a filling defect in the left renal pelvis, a finding which was confirmed by retrograde pyelography (Fig. 3, *A*). Left nephrectomy was performed on May 11, 1951, confirming the diagnosis of papillary carcinoma of the renal pelvis (*Fig. 3, C*). In the belief that the pulmonary lesion represented an isolated, synchronous metastasis, a wedge resection of the mass was accomplished at thoracotomy on June 4, 1951. The tumor was immediately sent for frozen section examination The pathologist reported that the lesion, although an epidermoid carcinoma, failed to resemble the renal lesion in degree of differentiation, or in architecture, and was most likely of bronchial origin, an opinion subsequently confirmed by the permanent sections

C.





D.

Fig 3 (*Cont'd*)

C, Photomicrograph of the renal tumor. The papillary nature of the lesion is demonstrated

D, Photomicrograph of pulmonary lesion

(Fig. 3, *A*). On the basis of this finding, the patient was returned to surgery five days later, and a left pneumonectomy was performed. The patient made an uneventful recovery, but succumbed eight months later in congestive heart failure, without clinical evidence of recurrence of either malignancy. Even though the possibility that these two lesions represent a primary renal tumor and a synchronous pulmonary metastasis cannot be contested with finality, the opinion of the reviewing boards designated the pulmonary lesion as being distinct from the renal lesion. On the basis of the considered opinion, this patient is listed as harboring two distinct carcinomas.

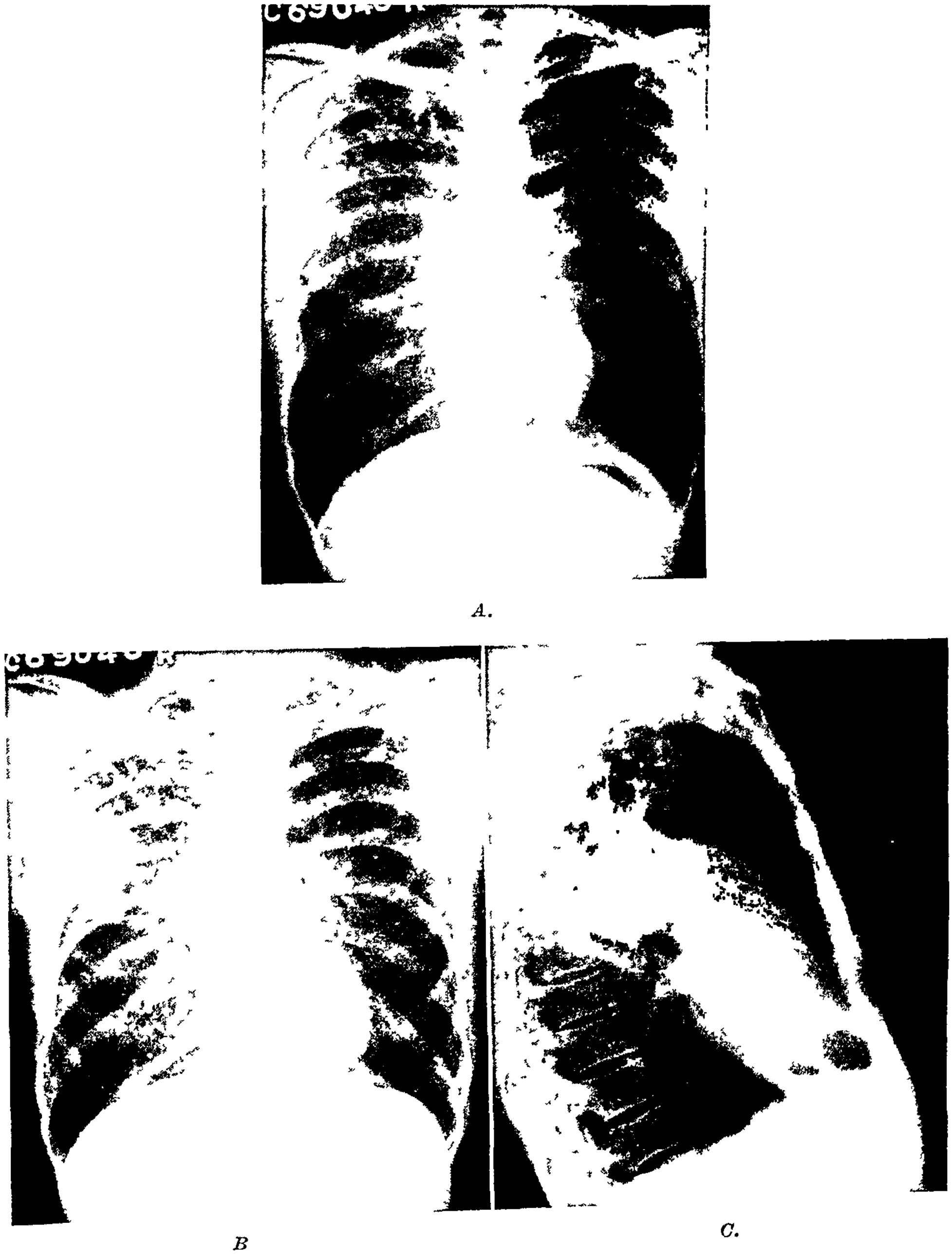


Fig. 4.—Case 4.

A, Chest x-ray of April, 1951, shows a small lesion overlying the right fourth anterior rib. The changes seen in this film were apparently interpreted as being due to pulmonary tuberculosis. Sputum studies were negative.

B, Chest x-ray of September, 1951, shows progression of the pulmonary lesion.

C, Chest x-ray in lateral view of *B*.

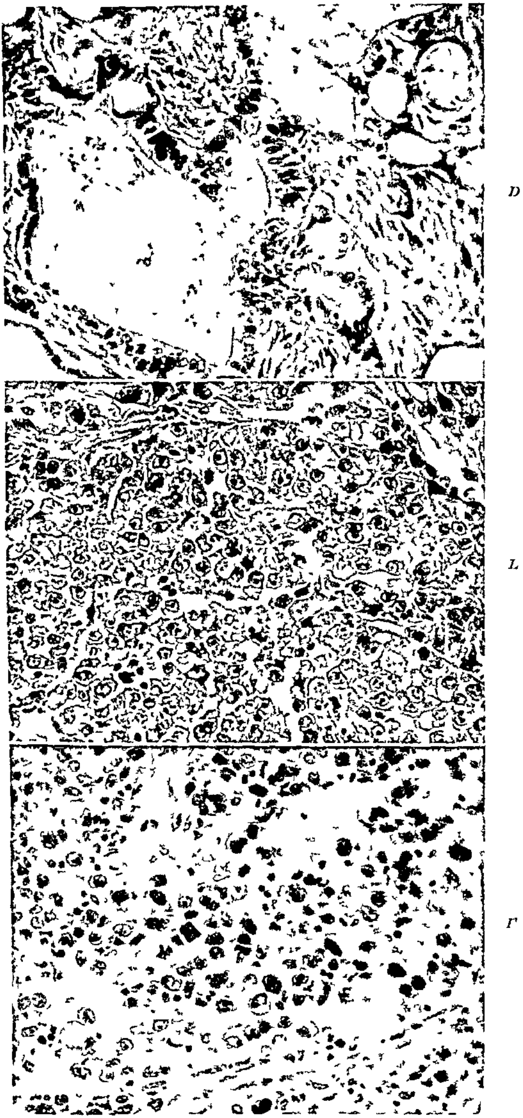
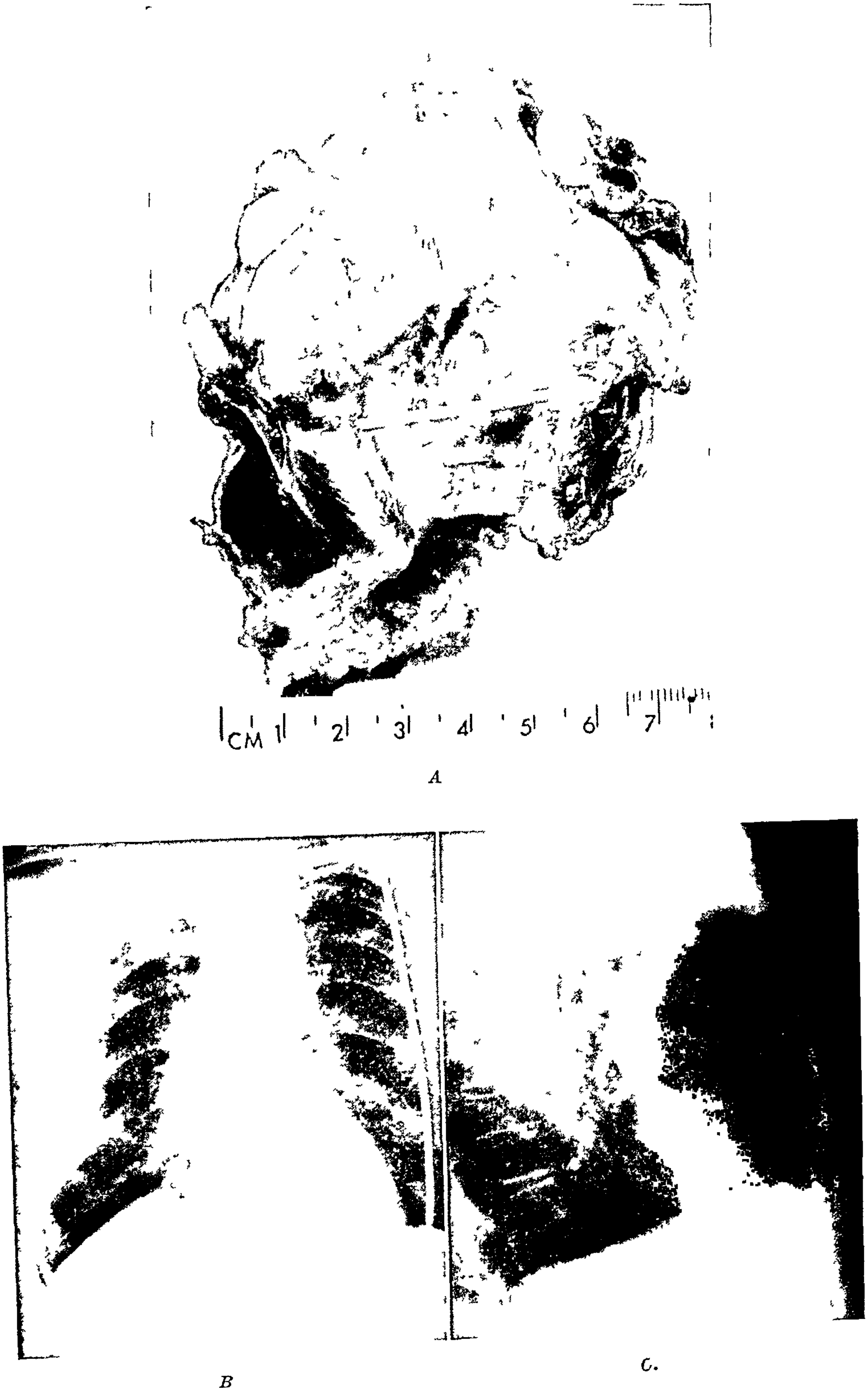


Fig 4 (*Cont d*)

D, Photomicrograph of gastric lesion showing typical adenocarcinoma

E Photomicrograph of pulmonary lesion showing broad sheets of pleomorphic epithelial cells No gland formation is seen

F, Section of right adrenal showing extensive infiltration by tumor cells similar to those seen in the pulmonary neoplasm



A

B

C.

Fig 5.—Case 5
A, Larynx, gross specimen Fungating lesion of both vocal cords is demonstrated.
B, Chest x-ray reveals a discrete peripheral lesion in the right upper lobe.
C, Chest x-ray in lateral view of *B*.

Case 4.—P. S., a 55 year old man, was readmitted to Hines Hospital on Aug. 21, 1951, with the complaint of persistent aching pain in the right posterior chest of two weeks' duration. He had been discharged from the hospital two months previously following a subtotal gastric resection for adenocarcinoma of the antrum (Fig. 4, D). Chest x-ray of April 24, 1951 (Fig. 4, A), prior to the gastric surgery, revealed scarring and fibrosis compatible with an old tuberculous process in the right apex. A small triangular density noted laterally at the level of the right fourth anterior rib was felt to represent a reactivation of the tuberculosis. Sputum smears and cultures were, however, negative on this admission. Chest film taken at the time of readmission showed marked increase in the size of this lesion, a rounded discrete density measuring 4.5 cm. in diameter now being visible in the base of the right upper lobe (Fig. 4, B and C). In the belief that this represented a

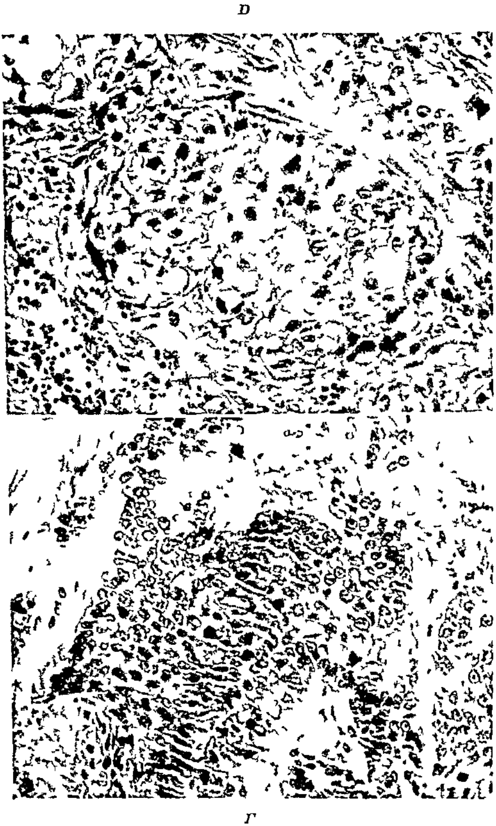


Fig. 5 (Cont'd).

D, Photomicrograph of a section of laryngeal lesion showing a typical squamous cell carcinoma.

E, Photomicrograph of pulmonary tumor showing again a typical squamous cell carcinoma.

right thoracotomy was carried out on Sept. 28, 1951. The lesion was found to cross the fissure involving the middle lobe and had extended into the chest wall at the level of the third and fourth lateral interspaces. A right upper and middle lobectomy were performed with considerable difficulty due to extensive vascular adhesions to the mediastinum and parietal pleura. Persistent oozing from these surfaces was encountered throughout the procedure which required four and a half hours for completion. Re-exploration was performed six hours later because of continuing hemorrhage, but proved futile, and the patient died four hours later. Histologic study of the pulmonary lesion showed a very anaplastic tumor made up of closely packed sheets of polyhedral cells, without any attempt at gland formation (Fig. 4, *E*). At autopsy no local persistence of either gastric or pulmonary lesion could be found. There was, however, almost complete replacement of the right adrenal by an undifferentiated tumor similar to that found in the lung, a circumstance which supported the diagnosis of bronchogenic carcinoma (Fig. 4, *F*).

CASE 5.—F. L., a 44-year-old man, was first admitted to Hines Hospital on July 20, 1953, with a diagnosis of tumor of the larynx. Past history revealed the onset of chronic cough and hoarseness fifteen months prior to admission. Direct laryngoscopy and biopsy were carried out a short time later and a diagnosis of carcinoma of the left true cord was made. He then received deep radiation therapy, consisting of 35 treatments (the factors of which are unknown) with complete subsidence of symptoms and disappearance of the lesion. Three months later, however, there was a recurrence of the tumor which was then treated by radium for a total of forty-one hours, again with good subjective and objective improvement. Five months before admission to Hines, a second recurrence appeared, and on his first examination here a fungating lesion was found involving both true cords with supraglottic and infraglottic extension. A total laryngectomy was performed on Aug. 13, 1953, without difficulty (Fig. 5, *A*). There was no evidence of extralaryngeal extension and the cervical nodes were free of involvement by gross and microscopic examinations. A preoperative chest x-ray on this patient had demonstrated a discrete lesion involving the right upper lobe of the lung (Fig. 5, *B* and *C*). Consideration of this lesion had been deferred until the laryngeal surgery could be accomplished. On Sept. 16, 1953, an exploratory thoracotomy was performed. A hard mass, rather centrally placed, was found in the right upper lobe. There was extension of tumor along the upper lobe bronchus to the tracheobronchial angle, with massive metastatic lymphadenopathy in the region of the superior pulmonary vein and posteriorly along the esophagus. The lesion was not resectable, and the chest was closed after biopsy. Histologic study showed both the laryngeal and pulmonary tumors to be squamous cell in origin and to be comparable in differentiation (Fig. 5, *D* and *E*). It was felt, however, that the absence of cervical lymph node involvement, the usual site of metastases from the laryngeal tumor, together with the finding of massive mediastinal and hilar lymph node involvement, undoubtedly secondary to the pulmonary lesion, supported the diagnosis of separate primary tumors.

DISCUSSION

It has long been known and accepted that, in most instances, the nature of a localized or rounded intrapulmonary density cannot be determined except by histopathologic section. When this circumstance occurs in an individual who has harbored or does at that time harbor a known neoplasm of malignant mien, the probability that the pulmonary lesion is likewise a neoplasm is undoubtedly increased. The inclination has been to consider the pulmonary lesion as representing a metastatic deposit from the original neoplasm because the lungs form such a predilect site for the deposition of such lesions.

The increased incidence of bronchogenic carcinoma, particularly in the male sex (our series is composed entirely of men), highlights the possibility that a second neoplasm occurring in a man would be of such nature. Even though metastatic lesions to the lung may lodge in such manner as to involve the bronchus, the classical roentgen picture is, however, that of a discrete parenchymal shadow. It is that select group that has interested us. Thus, we have interested ourselves only in the possible incidence of a bronchogenic carcinoma occurring under circumstances that would be most confused with a metastatic pulmonary lesion. There is no available means of determining whether such a lung lesion represents a metastasis or a new tumor, and at times this is so even when both lesions are subjected to microscopic examination because bronchogenic carcinoma can resemble tumors arising in other organs. From a clinical standpoint, to be able to decide this issue accurately would be very gratifying because it is suggested in our experience (Case 1) that metastatic tumors may remain localized for a reasonably long period of time even though the possibility of producing daughter metastases must be considered. If advantage were taken of such time, greater assurance might be had concerning the solitary nature of the metastatic involvement as well as further observations concerning the degree of control at the primary site, thereby avoiding some futile operation. On the other hand, we are hesitant to take this time for observation because there is no such "period of grace" in the event the lung lesion represents a second primary carcinoma. This is clearly illustrated in Case 2.

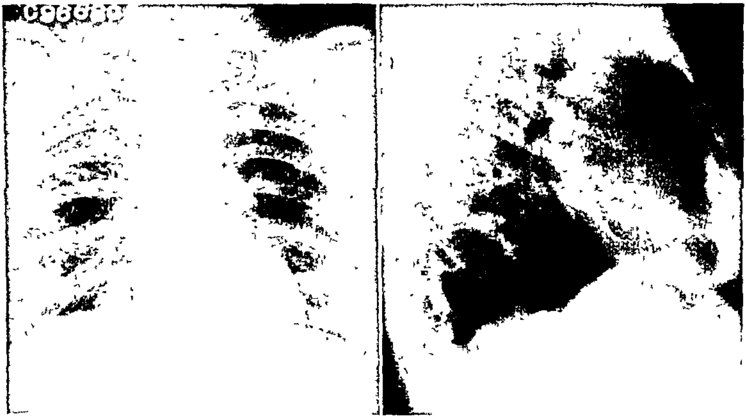

A. *B.*

Fig 6.—Case 6.
A, Chest x-ray reveals a discrete lesion overlying the right second anterior rib.
B, Chest x-ray in lateral view of *A*.

We, thus, are left with no alternative but to be bold in attacking discrete pulmonary densities under such circumstances. The following case serves to emphasize this point, since from a number of aspects the probability of a new pulmonary growth was suggested, yet the tumor proved to be metastatic.

CASE 6.—J. R., a 66-year-old man, was admitted on Oct. 8, 1953, for repair of a ventral hernia which had been present since 1945 when he underwent an anterior resection of the colon for obstructing carcinoma. Since this surgery, the patient had been followed at intervals of six months and had shown no evidence of recurrence. Physical examination was entirely negative except for a large, left-sided incisional hernia. A chest x-ray, however, showed a discrete lesion in the right upper lobe (Fig. 6, *A* and *B*). Because of the long tumor-free interval, the absence of other metastases, and slight prominence of the upper portion of the right hilum, a tentative diagnosis of bronchogenic carcinoma was made. Bronchoscopy was nonrevealing. On Oct. 23, 1953, a right thoracotomy was performed. The tumor was centrally located, the hilum was free from any involvement and a right upper lobectomy was accomplished without difficulty. Histologic sections then established the lesion as being unmistakably metastatic in origin (Fig. 6, *C* and *D*).

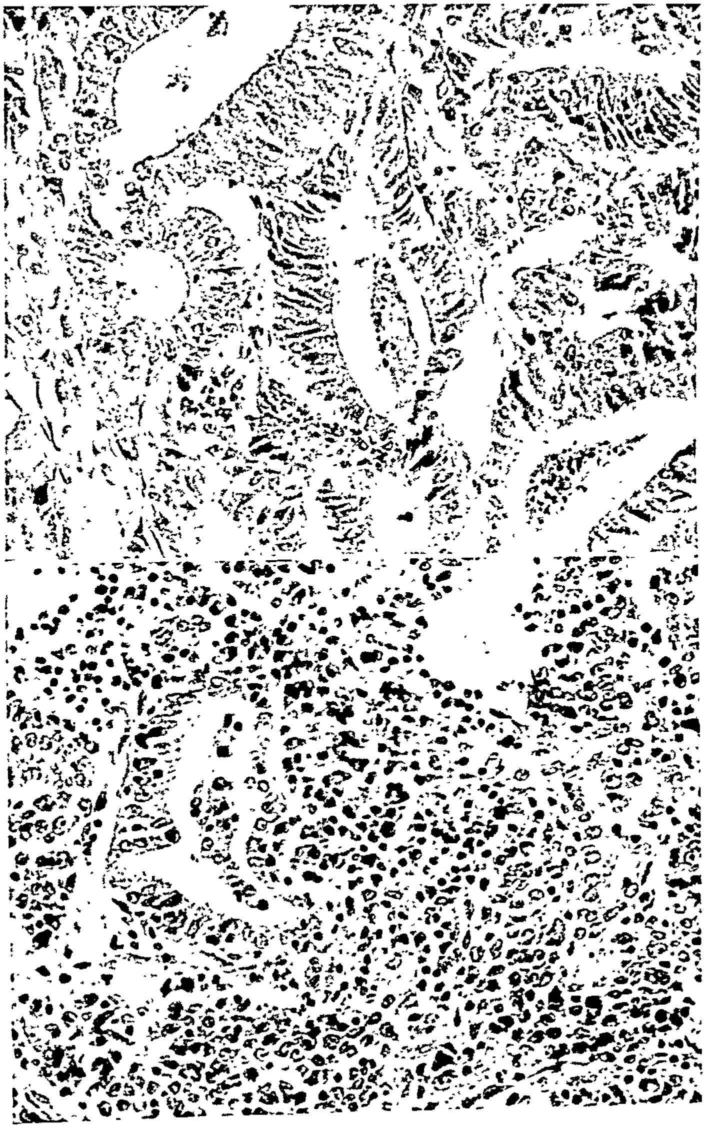


Fig. 6. (*Cont'd*).

C, Photomicrograph of the colon lesion showing a typical adenocarcinoma of the sigmoid.

D, Photomicrograph of the pulmonary lesion. This shows a less well differentiated tumor when compared to the primary in the colon but the rather characteristic gland formation denotes its colonic origin.

PLASTIC REPAIR OF ESOPHAGEAL FISTULA OF TUBERCULOUS ORIGIN WITH A DIAPHRAGMATIC CUFF

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FISTULA of the esophagus, due to caseation of the lymphatic glands, has appeared hitherto only in autopsy material. Among 7,000 autopsies, Vimtrup found 1,600 cases of tuberculosis, of which only 2 cases were of the esophagus. On investigation of 100 autopsies of tuberculous children, Sternberg discovered perforations in two cases only.

In half of all cases, esophageal tuberculosis seems to have its origin in the caseous degeneration of the lymphatic glands in the area of the hilus, and is therefore localized in the middle third of the esophagus. The appearance of tuberculosis in the upper third of the esophagus is less common. It is most infrequent, according to Hackel, who found it in only 12 per cent, in the inferior third.

Hackel, Pfeiffer, and Nawratil tried to strengthen the suture of the esophageal wall, which lacked serosa and was inclined to break open, by a muscle flap taken from its surrounding. The idea was not new, but these experiments had hitherto been confined exclusively to the cervical part of the esophagus. For the graft the sternocleidomastoid muscle was generally used. They tried to approach the fistulas of the cervical esophagus area by way of a posterior ediastinotomy extrapleurally. According to Sauerbruch, the results were isillusioning.

A lecture delivered by Professor Petrowsky at the Academy of Budapest, which he reported replacing the lower part of the esophagus with a diahragmatic cuff, induced me to attempt to strengthen the suture of the esophgus with a pedicled flap taken from the diaphragm, following the surgical xcision of a tuberculous fistula in the lower third of the esophagus.

CASE HISTORY

The history of the patient is as follows: E. G. arrived at the First Surgical Clinic f Budapest on April 8, 1951. She had been operated upon six months before at a country ospital, and since that time the fistula in her abdomen had been suppurating.

The filling of the fistula with contrast substance held a surprise, for it could be early seen that the abdominal fistula communicated with the diaphragmatic part of the ophagus (Fig. 1). No penetration of the contrast substance into the fistula could be certained when the patient swallowed. X-ray of the esophagus showed no pathologic teration which could have explained the perforation.

The following information was obtained from the hospital where the previous operaon had been performed: "Operation took place Oct. 18, 1950. Preoperative radiography

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From Surgical Clinic I, Budapest, Hungary; Director: Prof. Dr. Sebestény.

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on a horizontal level showed an abscess cavity, the size of a small nut, in the right subphrenic region near the vertebral column with an air shadow above it.'' The abscess was opened by laparotomy, and penicillin was applied followed by drainage. It was noted that the site of the nut-sized abscess cavity was in the anterior medial part of the right subphrenic region.

In the search for the etiology of the abscess, a roentgenogram was made of the spinal column, which showed that the intervertebral spaces were intact. X-ray examination of the thorax was negative. Westergren rate was 38 per 56 mm. Esophagoscopy showed no fistula. Number of leukocytes was 7,900. Wassermann reaction was negative. The qualitative blood examination showed relative lymphocytosis.

First, conservative therapy was tried. During the following two weeks, a daily dose of 1 c.c. potassium permanganate solution was given, but as this treatment turned out to be ineffective, during the next two weeks the fistula was cauterized with silver nitrate. The fistula still persisted. Thereafter, every third day a blood transfusion was given, and 0.25 Gm. of streptomycin was injected into the fistula daily. After two weeks, the secretion was much reduced, but a roentgenogram showed the fistula unaltered, and the opening into the esophagus was still present.

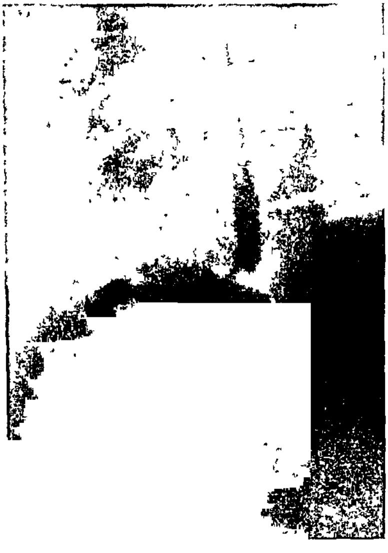

Fig. 1.—The communication of the abdominal fistula with the lower part of the esophagus was plainly demonstrated after the use of a contrast medium

Since the prolonged conservative therapy proved ineffective, an operation was decided upon. With preliminary pneumothorax and after adequate treatment of the heart, ether-narcosis was carried out with positive pressure produced by an apparatus after the method of Visnyevszkij with thoracolaparotomy. The fistula was reached by diaphragmotomy, and the right posterior wall of the diaphragmatic part of the esophagus was entered. After careful isolation, the fistula was excised from the esophagus and the lentilsized wound was sutured with interrupted sutures in two layers. We covered the esophagus with a 2-inch pedicled cuff-shaped flap, taken from the diaphragm at the level of the suture (Fig. 2).

The distal part of the fistula was resected, then the skin was closed by interrupted sutures. The diaphragm could be reconstructed satisfactorily. Using a thin Pezzer catheter in the thorax, the lungs were inflated and the thorax was closed. Through the Pezzer catheter, 200,000 units of penicillin was injected into the thorax. The abdomen was then closed and a thin rubber tube was introduced into the wound.

During the operation, 300 c.c. blood was administered by drop infusion. The patient withstood the operation well. During the first 24 hours, she received 40 c.c. of 10 per cent sodium chloride, 60 c.c. dextrose intravenously, 1.5 L. physiological sodium chloride subcutaneously and 2 L. rectally (Katzenstein). Penicillin was given parenterally, 40,000 units every 6 hours.

This therapy was continued for six days with a blood transfusion of 200 c.c. every second day. On the fifth day the patient received some bitter tea, a spoonful at a time, on the sixth day some soup, and on the eighth day pulpy food. During the first three days, the pleural secretion was removed through a Pezzer catheter 4 or 5 times daily, and 50,000 to 60,000 units of penicillin was injected into the thorax.

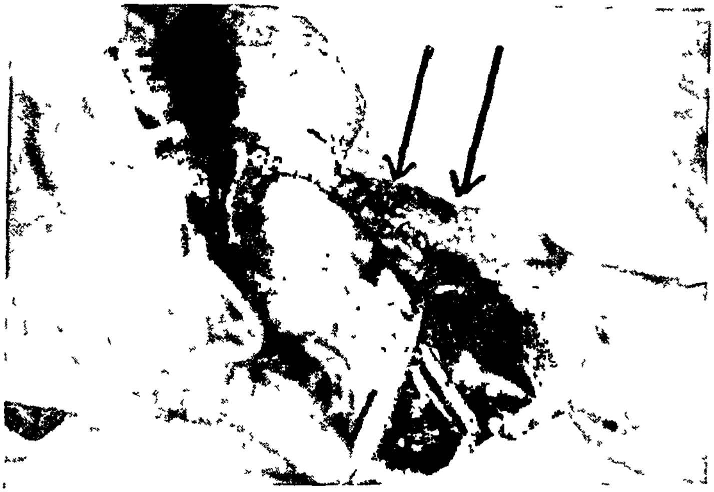

Fig. 2.—Between the two arrows the pedicled flap of diaphragm is seen about to be drawn over the fistulous opening of the esophagus

On the fifth day, radiography showed that the lung was entirely expanded and there was no secretion in the thorax, so that the Pezzer catheter could be removed. The abdominal fistula had been injected with 40,000 units of penicillin every second day, and the rubber tube also was removed on the fifth day. The fistula and the wound healed without any complications.

As the patient had had no history of a possible perforation caused by a fish bone or some other tiny foreign body, and as the whole clinical picture from the beginning indicated a specific cause, during the operation one of the pre-esophageal lymphatic glands was removed for biopsy. This showed *lymphadenitis chronica tuberculosa*. On the peritoneal side of the piece cut out from the diaphragm for histologic examination, tuberculous granulation was found as well. Since we found a necrotic lymphatic gland penetrating into the esophagus and the histologic finding verified our suspicion, the etiology of the process was judged to be tuberculous. The preoperative radiography, which showed an air shadow in the abscess cavity, also pointed to this, as the abscess had probably communicated with the esophagus. The operative opening of the abscess had completed the fistula.

The patient was examined postoperatively six months later. Her condition had improved a great deal. She had gained 6 kg. and was asymptomatic. Radiography showed that the contrast substance entered the stomach without hindrance. The patient was seen several times during the next four years. When last seen she was still without symptoms and was employed as a factory worker.

The case reported indicates that a diaphragmatic cuff is, in suitable cases, a reliable method of closing fistulas of the inferior third of the esophagus. This operative method seems to give an effective and durable solution to a rare disease. Study of the available literature failed to show a similar case.

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ANAPHYLACTIC SHOCK FROM INTRAPLEURAL STREPTOKINASE-STREPTODORNASE

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INTRODUCTION

STREPTOKINASE and streptodornase are proteolytic enzymes, derived from cultures of hemolytic streptococci, which are useful in enzymatic débridement. Streptokinase produces fibrinolysis by activating plasminogen (normally in human serum) to plasmin, which splits fibrin causing dissolution of blood clots and fibrinous exudates. Streptodornase liquefies desoxyribose nucleoprotein, which may constitute 30 to 70 per cent of purulent exudates. These enzymes are marketed commercially by Lederle Laboratories as Varidase. They have proved very useful intrapleurally as "biologic curettes" in treating clotted hemothorax and certain empyemas.

The following case is reported because of the unusual serious toxic reaction following its use:

CASE REPORT

L. J. Z. (St. Dominic's, 54-23904) was admitted to the hospital on June 18, 1954. Thirteen days previously he had sustained lacerations involving the left upper extremity, the right shoulder, and the right chest. There were two ribs exposed. The lacerations had been sutured in a doctor's office. Four days later he began to run a low-grade fever which spiked progressively higher and was associated with chills and with pain in the right chest and shoulder. On admission the patient appeared acutely ill. The blood pressure was 124/80; the temperature was 101° F.; the pulse was 88; respirations 24. The lacerations had healed well. There were dullness and decreased breath sounds in the lower right hemithorax. Chest film showed extensive haziness of the lower two-thirds of the right lung field. The patient had Grade II superficial varicosities of the lower extremities.

Multiple attempts at thoracentesis were done yielding only 40 c.c. of clotted blood. It was obvious that the patient had an infected, clotted hemothorax.

The following day 100,000 units of streptokinase and 25,000 units of streptodornase were injected into the right pleural cavity after aspirating with difficulty 60 c.c. of old blood. Twenty-four hours later 1,000 c.c. of serosanguineous fluid was removed; 400 c.c. was removed the following day. The gratifying response to this therapy led to its employment on the fourth day after admission. Following the second instillation, the patient had elevation of temperature to 104° F. and had some generalized itching. Following the second instillation of intrapleural Varidase, 1,500 c.c. of fluid was withdrawn.

On the eleventh day following admission a third intrapleural administration of Varidase was given. Four hours later the patient presented generalized giant urticaria followed by elevation of the temperature to 106° F. He then passed rapidly into a state of profound shock. Blood pressure was not obtainable. Vigorous treatment with anti-

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From the Surgical Clinic, Jackson, Miss.
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histaminics, oxygen, pressor agents, including l-arterenol* intravenously, cortisone (and later ACTH) was instituted. On continued infusions with l-arterenol, his blood pressure was maintained at about 70/40. Chest film revealed no evidence of fluid accumulation in the right pleural cavity or mediastinal shift (Fig. 1). Physical examination by several observers concurred in this. The l-arterenol infusions had to be maintained for three days before spontaneous circulatory stability was evident. During administration of the l-arterenol in the saphenous veins on both ankles by "cut downs" with polyethylene tubing, there was some extravasation of the vasopressor agent with resultant slough of the skin above the medial malleoli. Débridement and skin grafting of these areas were later necessary.

On July 6, 1954, 250 c.c. of straw-colored fluid was withdrawn from the right pleural cavity. Following this there was complete clearing of the right pleural cavity. He is now well and asymptomatic nine months after this severe anaphylactic reaction. He emphatically will not submit to skin testing to check on intradermal sensitivity to Varidase.

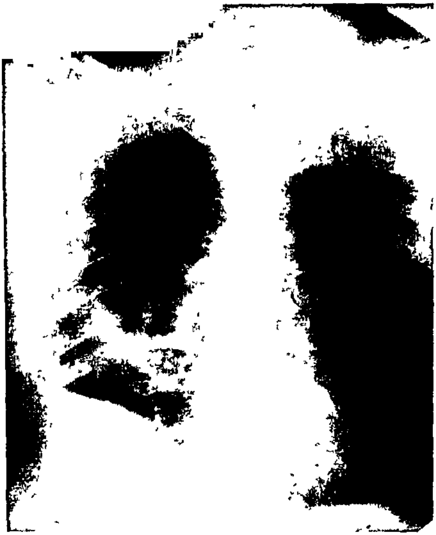

Fig. 1.—Portable chest film taken during period of anaphylactic shock showing no significant mediastinal shift.

DISCUSSION

Varidase acts by liquefying fibrin and nucleoprotein, thus facilitating the easier removal of clotted blood from the pleural cavity. Frequent aspirations are advised to provide good drainage.

Minor reactions may occur and are usually pyrogenic, following within four to six hours after the administration of the drug. Malaise, headache, and nausea are present in about 50 per cent of the patients.

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*Levophed, Winthrop-Stearns, Inc.

In 1952, a fatality following the intrathecal use of Varidase in a patient with tuberculous meningitis was reported.[2] Baum and Oransky[1] reported a case of circulatory collapse following the initial instillation of streptokinase and streptodornase in the pleural cavity. This patient was greatly benefited by the aspiration of 2,300 c.c. of fluid from the right pleural cavity, and an additional 1,500 c.c. was withdrawn the same day. The authors felt that this was an instance of anaphylactic shock. However, Miller and Long[4] in discussion of this case, disagree with this assumption, pointing out that the difficulty was caused largely by the rapid accumulation of pleural fluid with resultant mediastinal shift and cardiorespiratory embarrassment. They advised intercostal catheter drainage with connection to a Stedman suction pump to provide more adequate drainage of the pleural cavity in all but minimal instances of hemothorax. Miller and Long[4] stated that despite a very wide experience in the use of Varidase intrapleurally they had encountered no allergic or anaphylactoid responses.

Goehring and Grant[3] report a case which they interpret as anaphylactoid reaction following the use of a single intrapleural injection of streptokinase-streptodornase. A few hours later, the patient complained of chest pain and dyspnea; the blood pressure dropped to 70/40 mm. Hg and the pulse was rapid and thready. His condition remained critical until the next day when 600 c.c. of dark blood was removed by left thoracentesis. Chest film the following day showed "considerable increase in the amount of fluid in the left side of the chest. . . . The mediastinum and heart were displaced to the right." Skin test with streptokinase-streptodornase five weeks later was markedly positive with severe edema and redness of the entire flexor surface of the forearm and axillary adenitis. Although this points to sensitivity, it is not known whether this existed at the time of the serious initial reaction. Thus, it is easily possible that this shocklike state was not due to anaphylaxis but to mediastinal shift due to an increasing pleural effusion.

There can be little doubt that the case recorded here represents one of true anaphylactic shock resulting from sensitivity to streptokinase-streptodornase. Repeated physical examination and one by x-ray showed no evidence of significant pleural effusion or mediastinal shift. Thoracentesis was not done during the severe reaction and had nothing to do with the successful outcome. In retrospect, it is obvious that too little attention was paid to the generalized pruritus experienced after the second injection of drug; this was probably a lesser allergic phenomenon.

CONCLUSION

The case of a severe, fulminating, near-fatal anaphylactic reaction following the third intrapleural injection of streptokinase-streptodornase is reported to emphasize the definite antigenicity of the drug, which apparently increases with repeated injections.

The use of intradermal testing should be further investigated to evaluate its usefulness.

Careful search for minor allergic manifestations such as urticaria and pruritus should be made after each injection of the drug so that it can be promptly discontinued when they occur.

Prompt evacuation of the intrapleural products of liquefaction is urged by the use of intercostal catheter drainage as suggested by Miller and Long.[4]

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IMMEDIATE TREATMENT OF BRONCHOPLEURAL-PERITONEAL FISTULA

A Complication of Subphrenic Abscess

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PERFORATION of a subphrenic abscess through the diaphragm with the formation of a bronchopleural-peritoneal fistula was formerly a frequent complication.[1, 2] The sudden appearance of this additional burden in a patient already acutely ill from a subphrenic abscess places him in a precarious condition. The incidence and high mortality of this serious event have been greatly reduced by the employment of earlier and more adequate surgery, and the antibiotics. Immediate and well-planned procedures in correct sequence may initiate a rapid and gratifying improvement, as the following experience with 2 of these cases illustrates.

CASE REPORTS

Case 1.—L. T., a man, 44 years of age, underwent a gastric resection for duodenal ulcer at Swedish-American Hospital, Rockford, Illinois, on Dec. 6, 1952. Recovery was delayed by the development of a right subphrenic abscess which was drained by the conventional posterior approach (Ochsner[1]) on Dec. 22, 1952. The patient's temperature returned to normal on Dec. 28, 1952, and remained so until Jan. 8, 1953, when irregularly elevated temperatures reappeared. Drainage from the posterior wound had become intermittent, though at times it was quite profuse. On Feb. 1, 1953, the patient expectorated increasing amounts of a brownish sputum containing chlorophyll, which was also contained in a preparation being used to irrigate the posterior wound. He complained of pain in the right lower chest, and severe dyspnea was present. Markedly diminished breath sounds over the right side of the chest were noted. An x-ray of the chest showed extensive infiltration of the lower portion of the right lung, and a similar though less severe process on the left side (Fig. 1). The fluid level previously noted below the right diaphragm had disappeared.

He was first seen by us on Feb. 2, 1953. Extreme lethargy, weakness, and moderate disorientation were evident. Cyanosis was pronounced; respirations, 48 per minute, were shallow and noisy; and the pulse was 120 per minute and thready. Blood pressure was 86/60. Bronchoscopic aspiration was immediately performed with a little improvement which was not long maintained. The procedure was repeated two hours later, followed by a tracheotomy with the bronchoscope in the trachea. Tracheal aspirations were then repeated as indicated. On Feb. 3, 1953, he was deemed sufficiently improved to permit return to the operating room for drainage of the subphrenic abscess by the anterior subcostal route. Fourteen days later the bilateral pneumonitis had entirely cleared. Eighteen days later, on Feb. 20, 1953, he was discharged from the hospital. His subsequent convalescence was without incident.

Case 2.—J. V., an 18-year-old man, was admitted to Swedish-American Hospital for an appendectomy, which was performed on Jan. 24, 1952. Following operation a progressive temperature elevation was noted. A frank fecal fistula developed on Feb. 2, 1952. In the next few days this gradually closed. The patient's temperature remained high. An x-ray of the chest on Feb. 9, 1952, disclosed a right subphrenic abscess. At 9:45 P.M. on Feb. 9, 1952, the patient suddenly coughed up a huge amount of bloody malodorous sputum.

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Consultation was requested and the patient was seen by us at 10 45 P M His temperature was 104° F, pulse was 140 per minute and labored Restlessness, apprehension, and a grayish pallor were noted A foul odor was evident even at some distance from his room

Fig 1—Case 1, L T

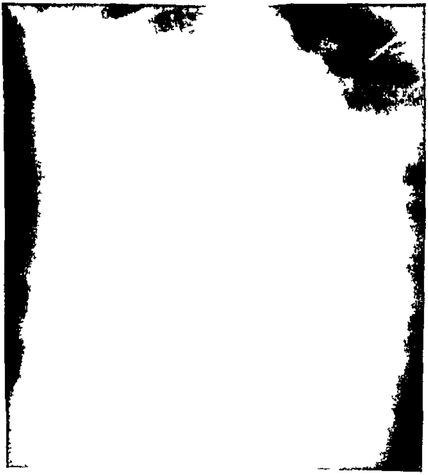

Fig 2—Case 2 J \

While preparations for operation were being hastened, respirations suddenly ceased and blood pressure and pulse became unobtainable. Resuscitation was achieved after endotracheal intubation. Drainage of the subphrenic abscess by the anterior subcostal approach, bronchoscopy, and tracheotomy were then proceeded with in that order. A large amount of pus was evacuated. Following surgery, the patient's pulse remained rapid and thready, respirations were shallow, and return to consciousness was delayed. A postoperative x-ray showed a massive infiltration of the right lung field with a similar though less extensive process on the left side (Fig. 2). After the first postoperative day recovery was uneventful. Final clearing of the pneumonitis was evident on Feb. 25, 1952. He was discharged from the hospital on March 2, 1952. Subsequent recovery was complete.

DISCUSSION

Improvement occurred in both these cases once emergency therapy was completed. Both patients would have benefited had there been no delay in the application of the several steps employed in the treatment. A bronchopleuroperitoneal fistula occurs in patients with a subphrenic abscess which has not been drained or has been inadequately drained. Its deleterious effects on the patient result from the sudden flooding of the respiratory tree with a large amount of pus. Treatment is properly directed at the removal of this material from above by means of aspirations by way of bronchoscopy and tracheotomy, and from below by adequate drainage of the subphrenic abscess. In the first patient, L. T., there was delay of two hours between the initial bronchoscopy and tracheotomy, and eighteen hours between tracheotomy and drainage of this abscess. In retrospect, it is felt that little was gained by deferring the drainage for that length of time. In the second patient, J. V., bronchoscopy and tracheotomy were delayed while preparations were made to do all three procedures at the same session in the operating room. Bronchoscopy and tracheotomy should have been performed immediately. A fatality nearly resulted from this error.

Local anesthesia is usually preferred for all indicated surgical procedures on these patients. The tracheotomy may be maintained indefinitely, since it greatly assists and accelerates the frequent clearing of the trachea and bronchi which is desired during the first few postoperative days. It also promotes a more effective pulmonary ventilation in these acutely ill individuals.[3] Positioning of the patient to facilitate drainage from below is of importance.

CONCLUSIONS

The sudden appearance of a bronchopleural-peritoneal fistula in a patient with a known or suspected subphrenic abscess presents a situation which calls for these procedures: (1) immediate bronchoscopic aspiration, followed directly by (2) tracheotomy, which in turn should be followed as soon as possible by (3) adequate drainage of the subphrenic abscess.

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CONGENITAL ANEURYSM OF THE SUPERIOR VENA CAVA

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SPONTANEOUS venous aneurysms are of rare occurrence. Two cases, located in the superior mediastinum, have been reported in the literature.[1, 2] An additional case with successful surgical excision constitutes this report.

CASE REPORT

B. D. (No. 02162), a 52-year old housewife, entered Barnes Hospital on March 5, 1953, complaining of pain located along the thoracic vertebrae. This had occurred over the previous two years and was associated with increasing exertional dyspnea. There was no history of chest trauma or previous surgery. An outside x ray was reported as showing a mass in the mediastinum, extending into the right lung field.

Physical examination, electrocardiogram, and laboratory data were noncontributory. An x-ray revealed a mass located in the right superior mediastinum which pulsated minimally during fluoroscopy (Fig. 1).

Fig. 1—Preoperative chest x-ray showing the aneurysm in the right mediastinum

Operation was performed on March 9, 1953. Under endotracheal gas oxygen-ether anesthesia a right posterolateral thoracotomy was made through the bed of the fifth rib which was excised subperiosteally. Covered by mediastinal pleura, above the azygos vein and lying between the superior vena cava and the vertebral bodies, was a blue cystic mass approximately 5 cm. in diameter (Fig. 2). On compression the cyst disappeared, only to re expand when pressure was released. Through its thin wall turbulent blood flow could be

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visualized. Needle aspiration yielded venous blood. The true nature of the aneurysm was elicited by dissection. The azygos vein was ligated and divided at the chest wall posterolaterally. The phrenic nerve was dissected from the anterior wall of the aneurysm which was then freed from the mediastinal structures medially. The aneurysm was seen to have a narrow, 2 mm. stalk at its point of origin, immediately above the junction of the azygos vein and superior vena cava. This was ligated and divided at the vena cava, as was the azygos vein. The chest was closed in the routine manner and the patient was discharged on the eighth postoperative day. When last seen on June 12, 1953, she was asymptomatic. A letter received in October, 1954, reported no dyspnea, but she complained of lumbar back pain. X-ray examination of the chest has revealed no recurrence of the aneurysm.

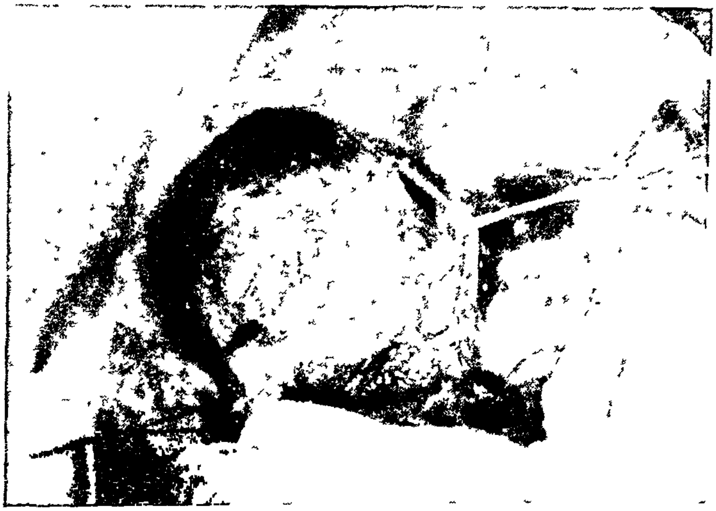

Fig. 2.—The appearance of the aneurysm at operation. The phrenic nerve is retracted anteriorly by the white tape and the azygos vein has been looped posterolaterally by the black ligature. The lung has been retracted inferiorly.

COMMENT

The case reported by Harris[1] occurred in a five-month-old infant who experienced spasmodic cough and minimal cyanosis. At post mortem the aneurysm was located in the right superior mediastinum and appeared to have communication with the left innominate, inferior thyroid, and right jugular veins. Abbot's[2] case was asymptomatic. First seen on a survey chest film, it was diagnosed, prior to operation, by angiography. This aneurysm appeared to arise from the superior vena cava. It was treated by cellophane wrapping of the vena cava and innominate veins. The patient, a 17-year-old student, did well postoperatively.

The present case illustrated the feasibility of surgical excision by ligation and interruption of the narrow communication with the parent vein.

SUMMARY

A case of successful surgical excision of a spontaneous or congenital aneurysm of the superior vena cava is reported.

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HEMOPTYSIS IN CANCER OF THE THYROID

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TWO interesting and unusual cases of a similar nature recently came to our attention In both there was marked hemoptysis of unknown origin, which later proved to be secondary to invasive but clinically inapparent carcinoma of the thyroid gland The seeming rarity of this clinical picture prompted us to attempt to discover any further cases fulfilling these criteria, here or elsewhere For this purpose, we reviewed the English language literature of the last twenty years and surveyed the records of 558 consecutive Mayo Clinic cases of carcinoma of the thyroid encountered in the 12 year period of 1940 through 1951

This study was made, first of all, in the hope that some common factors of a diagnostic import might be discovered, and secondarily, to ascertain, in so far as possible, the prognostic and therapeutic significance of invasion, in the absence of any other signposts of malignancy In our review we used the single factor of hemoptysis as the basis for selection of cases We did this for two reasons 1 The appearance of pure blood or red streaked sputum is sufficiently alarming to both patient and physician to guarantee its observance when present 2 Hemoptysis provides more definite evidence of true mural invasion than do such symptoms or signs as hoarseness, cough, and dyspnea all of which can be produced by external pressure on the trachea, or by involvement of the recurrent nerve, in the absence of any actual invasion

SEARCH OF THE LITERATURE

The literature on the thyroid is noticeably reticent on this subject There is no scarcity of cases in which a malignant lesion of the thyroid eroded through the adjacent walls of the larynx and trachea to appear within these structures Such an event, however, is invariably seen in cases of established carcinoma of the thyroid untreated or far advanced, in which the site of origin is only too clear In this group we find those tragic cases in which there is widespread invasion at the time of the patient's first visit to a doctor, and those cases in which there have been recurrences following palliative or subtotal resection for the neoplasm These cases are almost always terminal in nature In contra

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distinction, the true nature of our 2 cases was not evident initially, the underlying disease first manifesting itself solely through the facet of tracheal extension and intraluminal bleeding.

Search of the literature revealed only a very few cases in which the hemoptysis appeared at the time of the initial diagnosis of carcinoma of the thyroid. Frazell and Foote[1] reported one "bizarre" case in which the lesion first presented in the larynx; they did not elaborate. Cavenagh and McMenemey[2] recorded a case in which bloody mucoid sputum was produced but in which there was no evidence of invasion of the tumor at the time of operation. Grimes and Bell[3] reported 4 cases in which erosion of the larynx and hemoptysis occurred. In all of their cases, the thyroid was enlarged and stony hard, and the diagnosis was made preoperatively. They did note that an invasive tumor might not be seen at laryngoscopy or bronchoscopy in the presence of intact vocal cords.

MAYO CLINIC SERIES

In going over the records of the cases of thyroid cancer encountered at the Mayo Clinic during a 12-year period—558 cases in all—we were able to find only 17 cases in which the patient, at the time of initial examination and prior to any diagnostic or therapeutic procedure, gave a history of having had hemoptysis at any time in the past. The hemoptysis in these 17 cases preceded, followed, or became apparent at the same time as the appearance of the goiter in almost equal numbers of cases. This is of questionable significance, however, because of the known unreliability of any history regarding the duration of such a swelling.

The 2 cases that prompted this study are reported below.

CASE REPORTS

Case 1.—A 54-year-old white woman first noted hemoptysis, amounting to 1 teaspoonful of bloody mucus, in March, 1952, while attempting to clear her throat. She had undergone examination at the Mayo Clinic prior to this time, in January, 1952. At that time she had grade 1 diabetes mellitus and arteriosclerosis obliterans, but no respiratory or thyroid abnormality was observed. In November, 1952, when she returned after two more episodes of hemoptysis, the thyroid gland was clinically normal and no cause could be found for her hemoptysis. The results of laryngoscopic examination were negative. In April, 1953, the occasional gross hemoptysis had progressed to the point of daily expectoration of approximately a tablespoonful of bright red blood. She underwent bronchoscopy at that time, but nothing abnormal was noted.

In January, 1954, she came back because of the bleeding, which was always associated with coughing or clearing of the throat. This time bronchoscopy revealed an intratracheal tumor which bled very easily. Biopsy of this mass showed grade 1 papillary adenocarcinoma of thyroid origin. At exploration, the thyroid gland, when exposed, appeared to be quite normal. Only during the course of the ensuing right lobectomy was a small column of malignant tissue noted; this extended from the posterior surface of the lobe directly into the wall of the trachea. The trachea was split open in the mid-line, and the right half of it excised en bloc, the excision extending grossly wide of the tumor. A tracheal stoma was prepared by suturing the edges of a vertical skin incision to the remainder of the trachea, leaving it a shallow trough. The patient did quite well, and was able to go home two weeks after operation. When she returned to the Clinic at the end of April, 1954, she apparently was in good health. The trachea was repaired satisfactorily with auricular cartilage and local skin flaps.

Case 2 —A 71 year old woman had felt quite well until four years previously, when she first observed that if she happened to cough, as from inhaling too much dust, she always brought up blood streaked sputum This abnormality continued irregularly from that time forth Whenever she had a cold, she brought up some blood The hemoptysis increased in amount as time passed, and she became increasingly dyspneic and had inspiratory stridor When she was examined at the Mayo Clinic, the respiratory obstruction was quite obvious Three observers found no enlargement or nodularity of the thyroid gland Bronchoscopy, however, disclosed an intrinsic tracheal tumor that occluded the lumen Biopsy of the tumor revealed grade 1 papillary adenocarcinoma of thyroid origin, with some colloid formation Two days later, the patient underwent an operation on the thyroid At the time of exposure, the gland was recorded as being of normal size, but somewhat more nodular and firm than usual The site of the primary lesion could not be ascertained even then, and a subtotal left lobectomy was carried out first The neoplasm, however, was found subsequently when right sided lobectomy was performed, the tumor lay medially within the lobe and had eroded completely through the wall of the trachea No other invasions or local extensions were noted Following thyroidectomy, the trachea was split, and the invasive tumor and the surrounding tracheal wall were excised as a single block of tissue, a strip of trachea being left behind as framework for future reparative purposes There was no postoperative radiation and the patient went home, with a tracheal stoma, two weeks after operation Tracheal reconstruction has since been started according to the method used in the first case

One of these patients exhibited hemoptysis for two years and the other for four years before definitive treatment was instituted During these periods there had been no evidence of metastasis In each case the tumor was a low grade papillary adenocarcinoma We cannot as yet judge the ultimate prognosis because of the short lapse of time since operation

The importance of these cases is twofold (1) they point up a clinical picture that is seldom seen but one that the clinician should be aware of and (2) they suggest that good surgical treatment of cancer may be possible at times in cases in which at first the condition seems inoperable In fact, in both of our cases, complete removal of all apparent gross cancer was carried out in the definitive operation

The patient in Case 2 subsequently died after closure of the tracheal stoma Necropsy disclosed recurrence of carcinoma about the site of reconstruction

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SURGICAL CLOSURE OF ATYPICAL PATENT DUCTUS ARTERIOSUS

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SURGERY for the typical patent ductus arteriosus is among the least hazardous and the most gratifying procedures in the field of cardiovascular surgery. It is one of the few instances of cardiovascular abnormality in which the physician can pronounce a complete cure. The correction of atypical patent ductus arteriosus, on the other hand, is among the most dangerous of all surgical procedures. The presence of pulmonary artery hypertension greatly increases the operative risk, and the development of a right-to-left shunt makes the surgical correction of the ductus extremely perilous.

The operative morbidity and mortality in atypical patent ductus arteriosus is high. Sudden, complete closure of the ductus in the presence of pulmonary artery hypertension results in a further rise in the pulmonary artery pressure with its consequent severe, deleterious effects on the heart. Realizing that sudden, complete closure of the ductus would be fraught with danger we decided that perhaps gradual occlusion of the ductus would permit a slow reduction in pulmonary artery pressure and thus allow the body time to readjust to the hemodynamic changes. Such an approach would reduce the risk of an abrupt rise in pulmonary artery pressure and thereby increase the chances for the success of surgery.

CASE REPORT

M. L., a 7-year-old white girl, was referred to St. Michael's Hospital on June 13, 1952. At the age of 6 months, the child was treated for pneumonia and at that time the mother was told that a heart murmur was present. Subsequent growth and development were normal. Frequent upper respiratory infections occurred during early childhood. Cyanosis had not been noted. Dyspnea on exertion had developed within the past year.

Physical examination revealed a normally-developed young girl in no acute distress. The pertinent physical findings were confined to the cardiovascular system. A loud, harsh systolic murmur was heard at the third intercostal space to the left of the sternum. A short diastolic murmur was heard by some observers but not by others. A systolic thrill was palpable to the left of the sternum. Blood pressures were: right arm 120/50; left arm 120/40; right leg 170/50; left leg 170/50. The lungs were clear to auscultation and percussion. There was no clubbing of the fingers or toes.

Fluoroscopic and x-ray examination of the chest (Fig. 1) showed a marked increase in the pulmonary vascular markings bilaterally. A hilar dance was present. The pulmonary artery was enlarged and bulging. Both the right and the left ventricles were enlarged. The left atrium was also enlarged.

The electrocardiogram revealed a neutral axis and did not show any significant change from the normal.

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Laboratory data included: red blood count, 4,000,000; hemoglobin, 10.4 Gm.; hematocrit 42 per cent.

Cardiac catheterization was performed on Oct. 1, 1952. The findings are summarized in Table I. There was evidence of pulmonary artery hypertension, right ventricular hypertension, and both right-to-left and left-to-right shunts. The catheter was passed through the ductus and down to the abdominal aorta.

TABLE I. CARDIAC CATHETERIZATION DATA BEFORE SURGERY AND TWO YEARS POSTOPERATIVELY

	PREOPERATIVE		POSTOPERATIVE	
	PRESSURE IN MM. HG	BLOOD OXYGEN CONTENT IN VOL. PER CENT	PRESSURE IN MM. HG	BLOOD OXYGEN CONTENT IN VOL. PER CENT
Superior vena cava		12.49		12.45
Inferior vena cava		13.20		13.25
Right atrium	5/0	11.64	5/0	13.28
Right ventricle	88/0	11.41	30/0	12.56
Main pulmonary artery	100/60	14.73	28/10	13.37
Abdominal aorta	140/50	15.78		
Femoral artery			140/80	17.13
Arterial oxygen capacity		17.82		17.25
Arterial oxyhemoglobin Saturation (in per cent)	88		99	

Surgery was performed on Nov. 12, 1952. A short, wide patent ductus arteriosus was isolated. Purse-string sutures were applied to the ductus at both its aortic and pulmonary artery attachments. The sutures were tightened so as to occlude 75 per cent of the original lumen of the ductus. The body of the ductus was then wrapped with twisted polyethylene sheeting that had been activated previously in a solution of diacetylphosphate. There was no evidence of cardiac embarrassment at the time of occlusion of the ductus.

A to-and-fro murmur could be heard on the day following surgery. Postoperative blood pressure was 116/40. An x-ray film of the chest taken on the fifth postoperative day showed a diminution in the vascular markings.

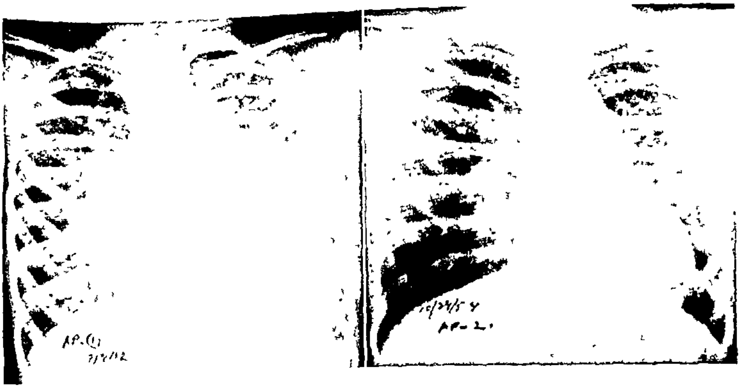

Fig. 1.—Thoracic roentgenograms demonstrating a diminution in the vascularity of the lung fields and a decrease in heart size 2 years after closure of an atypical ductus arteriosus.

The murmur, which assumed a machinelike quality, persisted until May 15, 1953, at which time only a harsh systolic murmur at the base could be heard. This murmur gradually diminished in intensity until Feb. 5, 1954, when no murmur could be heard.

X-ray examination of the chest on Oct. 28, 1954, revealed a diminution in the pulmonary vascular markings, and a reduction in heart size (Fig. 1).

The electrocardiogram at this time showed no essential change in comparison with the preoperative tracing.

Cardiac catheterization *performed on Oct. 28, 1954*, revealed normal pulmonary artery and right ventricular pressures. There was no evidence of either intracardiac or extracardiac shunt.

The child has been essentially asymptomatic since surgery.

DISCUSSION

The persistence of pulmonary artery hypertension and the development of a right-to-left shunt in atypical patent ductus arteriosus can only progress to the ultimate deterioration of the patient. A reduction in the pulmonary artery pressure and the elimination of the shunt would offer the individual a chance for survival and the hope of a normal life.

Johnson and associates[1] believe that most of the shunts in atypical patent ductus arteriosus are bidirectional and that the increased pulmonary blood flow doubtless plays a role in the production of pulmonary hypertension, and that ligation of the ductus would result in a marked reduction of both pulmonary blood flow and pulmonary hypertension. With these contributing factors reduced, it is conceivable that there could be at least partial regression of the pathological changes in the vessels and the pulmonary vascular resistance thereby decreased.

The success of surgery is related to the severity and reversibility of the pulmonary vascular pathology. The pulmonary hypertension in a large measure is a reflection of the pathological alterations in the medium-sized muscular pulmonary arteries. The essential changes are medial hypertrophy of the muscular coat and intimal fibrosis. These changes tend to narrow the effective lumen of the vessel and offer an obstruction to the pulmonary blood flow. Multiple vascular thromboses also have been described and add to the pulmonary vascular obstruction. The thromboses may be related to polycythemia.

The origin of the vascular aberrations is not known. Edwards[2] has offered the hypothesis that some patients with patent ductus arteriosus retain the fetal type of the medium-sized muscular pulmonary arteries. In expanding this viewpoint, Kattus and Muller[3] have stated that the presence of a large ductus more readily permits a transmission of the higher aortic pressure to the pulmonary arteries. The transmitted pressure then acts as a stimulus for the retention of the fetal type of vessels. Both have agreed that the intimal lesions can occur as a result of prolonged pulmonary hypertension. Lukas and associates[4] believe that the abnormally large flow through the ductus is the initiating factor in the development of pulmonary hypertension, but suggests that in some cases a retention of the fetal vasculature may play an important role, especially during infancy.

If the vascular changes regress following surgery, then improvement can be expected. If the changes have become fixed and irreversible little or no benefit could be expected from the surgery.

The results of surgery for patent ductus arteriosus with pulmonary hypertension have been disappointing. Crafoord[5] reported eight deaths among 16 patients with pulmonary hypertension who had been operated on for patent ductus arteriosus. When reversal of flow occurs the mortality is considerably higher. Table II summarizes the experience of several authors and the problems encountered at the time of operation. Difficulty arises from three main sources 1, the friable state of the ductus, 2, the development of cardiac embarrassment when the ductus is completely occluded, 3, the presence of a short, wide ductus

TABLE II EXPERIENCE OF OTHER AUTHORS IN THE SURGICAL CORRECTION OF ATYPICAL PATENT DUCTUS ARTERIOSUS

AUTHORS	OPERATION	RESULT
Dammann et al[6]	Occlusion of the ductus resulted in cardiac arrest	Patient expired
Hultgren et al[7]	Complete ligation	Doing well 2 months postoperatively
Kattus and Muller[3]	Complete closure	Developed right ventricle failure which did not respond to medical management
Bothwell et al[8]	Due to technical difficulties only a partial ligation could be performed	Doing well 2 months postoperatively
Holswade and Goldberg[9]	1 Hemorrhage during isolation of ductus 2 Shock with occlusion of ductus	Procedure abandoned Progressive failure Procedure abandoned
Pritchard et al[10]		Death eight hours after ligation of the ductus
Johnson et al[1]	Ductus closed by tightening two ligatures	Died fifth postoperative day following blood transfusion Autopsy ductus only partially ligated
Shumacker[11]	Tachycardia with application of clamp, after repeated clamping no effect, ductus then divided	Patient doing quite well
DeCamp[12]	Patient died 15 minutes after application of clamp	Patient died
Lukas et al[4]	1 Friable ductus with excessive bleeding 2 Obliteration of ductus caused a fall in blood pressure 3 Progressive deterioration of patients' cardiovascular status was not reversed by ligation of the ductus	Procedure abandoned Permanent ligation of ductus not attempted Patient died

Despite the poor operative prognosis, patients with atypical patent ductus arteriosus who survive the operation are usually benefited. There have been reports in the literature indicating that the pulmonary artery pressure can be reduced following surgery. Hultgren and co-workers have demonstrated a fall in pulmonary artery pressure from 110/62 mm Hg to 65/40 mm Hg one month after surgery. Adams[13] demonstrated a fall in the pulmonary artery systolic

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The changes in the pulmonary vessels are not immediately reversible. A period of months is required for regression to occur. Cournand and co-workers[15] were not able to show any change in pulmonary artery pressure five weeks following surgery. Similar studies performed five months postoperatively showed a normal pulmonary artery pressure.

It would appear, then, that surgery is indicated if the pulmonary vascular changes have not become irreversible and if the dangers of operation could be reduced. Complete occlusion of the ductus has resulted in a significant number of deaths. The friable ductus has caused technical difficulty requiring abandonment of the surgical procedure in several instances. The technique utilized in the case reported would appear to assist in solving these problems. The partial ligation of the ductus to 75 to 80 per cent of its diameter would tend to reduce the danger of a sudden and excessive rise in pulmonary artery pressure. The application of polyethylene sheeting permits the slow, gradual obliteration of the ductus. During the period of slow closure of the ductus, the body as a whole, and, in particular, the cardiovascular system, is permitted to adjust to the changing hemodynamics.

Had the ductus failed to close within eighteen months after the application of the polyethylene sheeting, surgery would have been performed to occlude the ductus completely.

An alternate procedure would be to ligate the ductus partially and then one year later a second-stage operation could be performed to occlude the ductus completely.

SUMMARY

A case of patent ductus arteriosus with pulmonary hypertension and bidirectional shunt is presented. Delayed closure of the ductus was accomplished by partial ligation and wrapping with polyethylene sheeting. The feasibility of surgical obliteration of atypical patent ductus arteriosus is discussed.

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LYMPHANGIOMA OF THE MEDIASTINUM

REPORT OF A CASE WITH REVIEW OF THE LITERATURE

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CYSTIC *lymphangioma is infrequently confined to the mediastinum.* As disclosed by the available literature, operative intervention has been practiced seventeen times. Our case brings the total to eighteen (Table I). Other references to these cysts included those found at autopsy,[10, 17, 18] those of doubtful diagnosis,[4, 5] and discussion of th ith no case presentation.[11, 14]
None of these references was considered the present
In presenting an additional case, th ers are an
attempt to clarify some of the problems c osis, and

EMBRY

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The possible relationships of these embryological findings to the origin and development of lymphangiomas will be discussed subsequently

CASE REPORT

History—H B, a 42 year old white man, was admitted to Samuel Merritt Hospital in Oakland, Calif, on April 4, 1950 He complained of dyspnea and mild substernal discomfort Throughout his life he had noted shortness of breath and weakness This did not prevent him from participating in athletics, but he was aware that his reserve had always been less than that of his playmates Three years before admission, he had been refused life insurance because of an increase in the severity of his symptoms and the finding of an "enlarged heart" Two to three pillow orthopnea, frequent colds, and bronchial wheezing followed In 1949, he developed an upper respiratory infection with sinusitis, cough, and greenish, mucoid, blood-streaked sputum

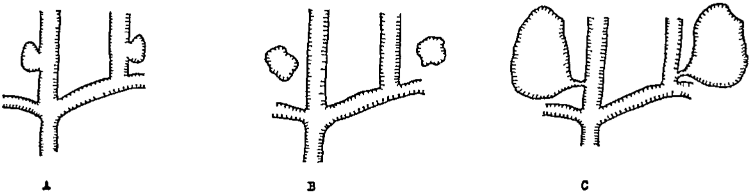

Fig 1—Stages in the formation of the primitive jugular lymphatic sacs (diagrammed from a description by Sabin[70]) *A*, Endothelial pouches formed from the internal jugular veins *B*, Lymphatic sacs divorced from internal jugular veins *C*, Paired jugular sacs reunited to the jugular veins

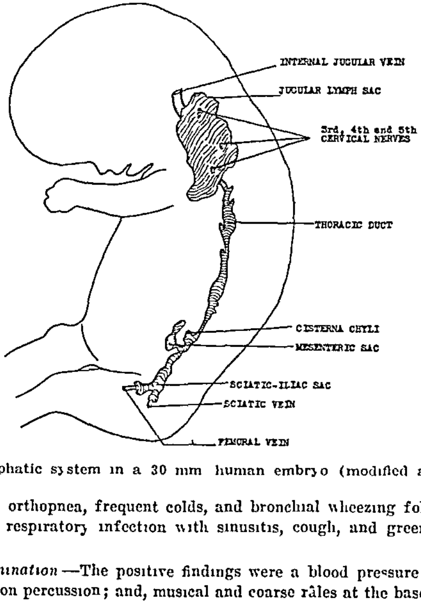

Fig 2—Lymphatic system in a 30 mm human embryo (modified after Sabin[70])

Physical Examination—The positive findings were a blood pressure of 158/80; a heart enlarged to the left on percussion; and, musical and coarse râles at the bases of both lungs and at the left apex.

ANGIOMAS OF THE MEDIASTINUM

CASE	AUTHOR	YEAR	AGE	SEX	THERAPY	ANATOMIC LOCATION	HISTOLOGY	CONTENTS	RESULT
1.	Skinner and Hobbs[26]	1934	7	M	Two-stage incomplete excision	Pericardium, superior vena cava, thymus	Fibrous wall with smooth muscle, lymphocytes, areas $\bar{c}$ cholesterol crystals; lined by endothelium	Thin, watery, brown fluid	Empyema. Well ten years postoperatively[25]
2.	Bumin[3]	1939	25	M	Excision	Diaphragm, pericardium	?	?	Improved
3.	Sanes, Macmanus, and Scatchard[22]	1943	59	M	X-ray Excision	Trachea, esophagus, azygos vein	Fibrous wall with smooth muscle, lymphocytes; lined with flat cells; hemosiderosis	Thin, watery, yellow-brown fluid	Paralysis right vocal cord. Well 11 months postoperatively
4.	Gross and Hurwitt[8]	1943	32	M	Excision	Anterior to right phrenic nerve from diaphragm to great vessels	Fibrous wall lined with endothelium	Clear amber fluid	Well three years postoperatively
5.	Watson and Diamond[29]	1947	24	M	Excision	Trachea, superior vena cava	?	?	Improved
6.	Santy, Galy, Jaubert de Beaujeu[23]	1947	25	F	Biopsy Drainage Aspirations	Right lateral tracheal wall	Fibrous wall with smooth muscle	Clear fluid	Cyst remains?
7.	Santy, Galy, Jaubert de Beaujeu[23]	1947	47	M	Incomplete excision	Left anterior chest wall, pericardium	Fibrous wall with islets of fat; lined by endothelium	Clear fluid	Recurrence of cyst three years postoperatively
8.	Emerson[6]	1947	20	F	Excision	Posterior to right pulmonary hilum	Fibrous wall with lymphocytes, smooth muscle; lined with elongated cells	Milky fluid	Improved three months postoperatively
9.	Steckler, Gordon, and Reisman[27]	1948	4	F	Incomplete excision	Lateral to inferior vena cava	Fibrous wall with islands of slitlike cholesterol and fatty acid crystals, foreign body reaction; lined with endothelium	Clear yellow-tinted fluid	Well immediately postoperatively

10	Harley and Drew[9]	1949	2	M	Excision	Pericardium, diaphragm sternum, 2nd to 4th intercostal spaces anteriorly on right	Fibrous wall with smooth muscle, lymphocytes, eosinophils, lined by endothelium	Watery straw colored fluid	Well immediately postoperatively
11	Skinner, Isbell, and Carr[24]	1949	54	M	Methyl bis X ray Excision	Superior vena cava, azygos vein, esophagus	Mesothelium lined cyst	Thin clear fluid	Well one year post operatively
12	Lindskog[15]	1950	?	?	Excision	Anterior costophrenic angle	Wall contained smooth muscle, mesothelium lining	?	?
13	Lindskog[15]	1950	?	?	Excision	Anterior costophrenic angle	Wall contained smooth muscle, mesothelium lining	?	?
14	Touroff and Seley[28]	1950	3	M	Incomplete excision Chemical sclerosis	Between lower esophagus, aorta, diaphragm, and pericardium	Hygroma	Milky fluid	Well two years postoperatively
15	Childress, Samson, and Baker (this report)	1950	42	M	Excision	Anterior to pericardium and great vessels from thoracic inlet to diaphragm, left phrenic nerve incorporated	Fibrous wall with smooth muscle, lymphocytes, lined by endothelium	Milky fluid	Paralysis left hemidiaphragm, wound infection, well five years post operatively
16	Balst[2]	1951	42	M	Excision	Right esophagophrenic angle	Fibrous wall with elastic and muscle tissue, no specific lining	Milky fluid	Well two months postoperatively
17	Bagolan, Alati and Fisicaro[1]	1953	15	F	Excision	Beneath left phrenic nerve, lateral to pericardium and anterior to ascending aorta	Fibrous wall with lymphocytes, lined with endothelium	Liquid	Well immediately postoperatively
18	Bagolan, Alati and Fisicaro[1]	1953	61	F	Excision	Trachea, esophagus, azygos vein	Fibrous wall with lymphocytes, lined with endothelium	Reddish brown fluid	Well immediately postoperatively

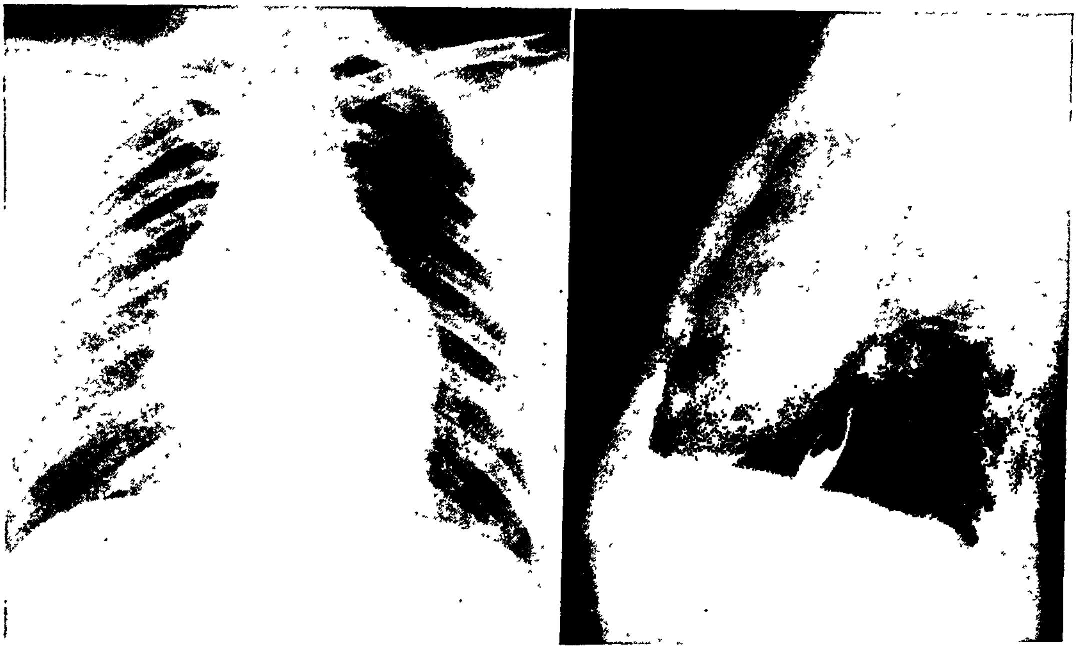

Fig. 3.—Posteroanterior and lateral roentgenograms demonstrating the anterior mediastinal shadow superimposed upon that of the heart and great vessels.

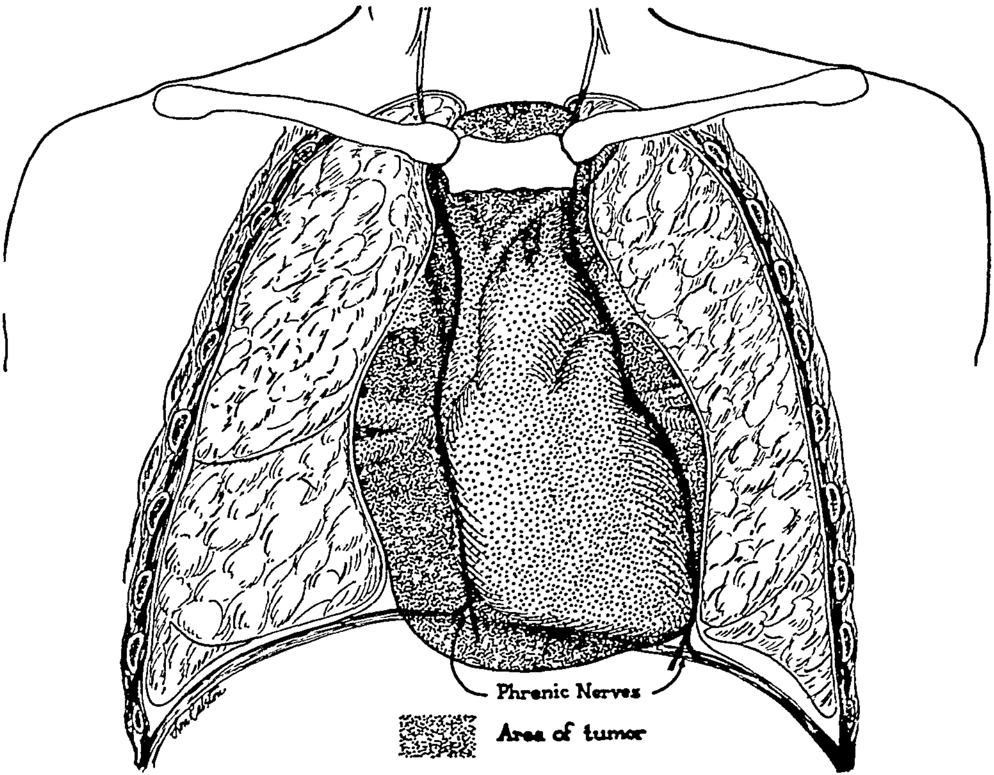

Fig. 4.—Extent of the cyst as depicted by the artist from a description.

Roentgenographic Examination.—There was an abnormal density extending from the thoracic inlet to the diaphragm. The density was thought to be primarily in the anterior mediastinum and to surround the heart. The borders of the mass were noted to pulsate slightly during fluoroscopic examination, but were interpreted as being transmitted from the cardiac impulse (Fig. 3).

Preoperative Diagnosis.—Anterior mediastinal tumor, probably a dermoid or thymoma.

Operation.—On April 5, 1950, a left exploratory thoracotomy was undertaken by one of us (P. C. S.). The chest was opened posterolaterally through the bed of the resected fifth rib. A pinkish-gray cystic structure filled the anterior mediastinum and extended from the thoracic inlet to the anterior cardiophrenic angle. Laterally, it covered the anterior aspect of left pulmonary hilum; the left phrenic nerve was incorporated in its substance. The cyst extended across the anterior mediastinum and overlapped the superior vena cava. Inferiorly, it spread over the pericardium to the diaphragm along both margins (Fig. 4). During extirpation, the left phrenic nerve was necessarily sacrificed. When the cyst was inadvertently entered, milky fluid with a pinkish tinge issued forth. Examination of this fluid and biopsy of the mass revealed it to be a lymphangioma. The right pleural cavity was then entered transmediastinally. By means of traction and dissection, it was possible to remove the remaining portions of the cyst from the superior vena cava and the right side of the pericardium. Great care had to be exercised to spare the right phrenic nerve. Grossly, the cyst appeared completely removed.

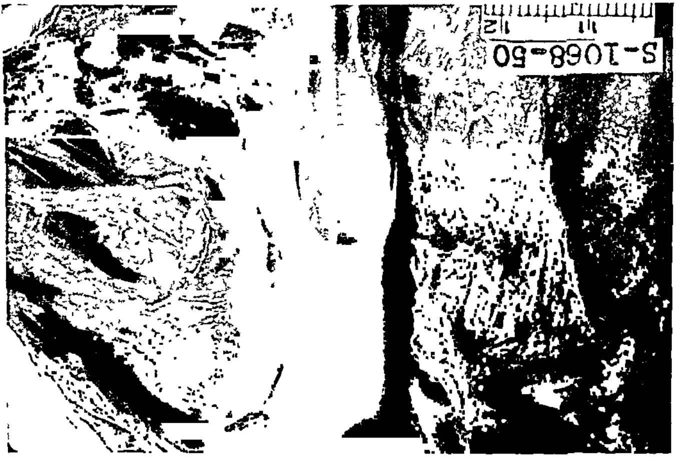

Fig. 5.—Lymphangioma with large spaces and fibromuscular trabeculae. Milklike fluid content in test tube.

Pathologic Report.—The tumor measured 16 by 10 by 5 cm. It contained milky fluid with a high lipid content. The outer wall was shaggy and pinkish gray. The inner surface was crossed by many thin, interlacing trabeculations and presented a smooth, shining surface (Fig. 5). Microscopic section showed an outer fibrous wall with an inner layer of flattened endothelium. The fibrous wall contained smooth muscle, collections of lymphocytes, adipose tissue, and blood vessels (Fig. 6).

Postoperative Course.—Wound infection was obvious by the third day and a nonhemolytic staphylococcus was cultured from the exudate. Five months following the thoracotomy, it was necessary to unroof several sinuses which communicated with the extrapleural space. Healing then proceeded uneventfully. The patient is now well and working, more than five years following excision of the lymphangioma.

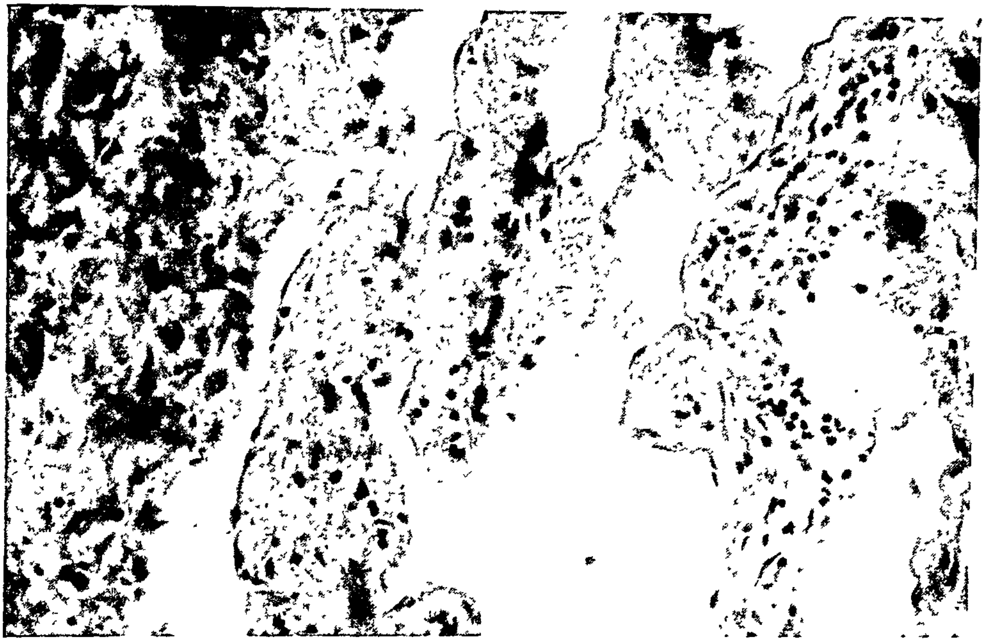

Fig. 6.—The totally irregular spaces are lined with a single flattened layer of endothelium. In the unsectioned tumor these spaces contained chylous fluid. The fibromuscular septa (trabeculae) and a portion of the cyst wall are shown. (×300.)

DISCUSSION

In a review of the 18 cases available to us, the age at diagnosis varied from 2 to 61 years. Eleven of these patients were men and 5, women. In 2 cases the age and sex were not tabulated. The symptoms were nonspecific but pointed, in general, to some intrathoracic disturbance. Cough was the most prominent, presumably due to bronchial or tracheal pressure. Expectoration, hemoptysis, dyspnea, and pain in the chest trailed in frequency. Poor growth and development in one child probably were related to a chylothorax.[28] Hoarseness due to paralysis of the *left* vocal cord occurred in 1 case with a cyst high in the right mediastinum.[24] This hoarseness persisted postoperatively and its cause remained obscure. It misguided the authors. On first reflection, they considered it a sign of inoperability for a lesion which subsequently proved to be operable.

The time interval between the onset of symptoms and discovery of the lesion varied from one month to many years. In at least one-half of the cases, symptoms persisted for more than one year. In 4 instances, however, the lesion within the thorax was unsuspected until disclosed by a roentgenogram taken for other reasons.

Preoperatively, the true nature of the roentgen shadow remained undetermined in all cases: thoracotomy was performed 17 times; cervical mediastinotomy was employed in a single case.[23] Diagnostic pneumothorax revealed an extrapulmonary tumor in two instances,[26, 27] but was unrewarding in a third.[28] Skinner and Hobbs[26] outlined the interior of the cyst by injecting air into it, for contrast with the diagnostic pneumothorax. Bronchoscopy, esophagoscopy, and esophagograms suggested extrinsic pressure in a few cases.

A test dose of x-ray was used twice to note its effect on the mediastinal mass. In one case, no change in size was noted.[22] In the other, the mass diminished and led the authors to the mistaken conclusion that they were dealing with a lymphoma.[24]

Emerson,[6] during thoracotomy, demonstrated a communication between the cyst and the thoracic duct. Lipiodol was injected into the cyst and a roentgenogram was taken on the operating table.

ANATOMIC LOCATION

The relationship of these tumors to the mediastinum is clear. One can readily visualize them expanding laterally from the less yielding mediastinal structures to the more compressible pulmonary tissue. Among the cases so documented, extension into the right hemithorax was noted 11 times, and into the left 4 times.

Certain patterns of location are discernible when one considers the 18 collected cases. Five[1, 22, 23, 24, 29] were directly related to the triangle formed by the superior vena cava, esophagus, and azygos vein. The anterior costophrenic angle and related structures housed eight.[1, 8, 9, 15, 23, 26] Of the remainder, one was posterior to the right pulmonary hilum,[6] one was in the right esophagophrenic angle,[2] one was lateral to the inferior vena cava,[27] one was between the lower esophagus-aorta-pericardium-diaphragm,[28] and the present case occupied the entire anterior mediastinum.

ORIGIN

Goetch[7] in a scholarly presentation concluded that portions of the primitive jugular sac may be sequestrated and fail to establish communication with the venous system; further, that the sequestrated part may continue to grow in a manner characteristic of the primitive lymphatic system.

Reference to the embryology of the lymphatic system shows that such an explanation is applicable to the stage in which the primitive jugular sac is divorced from its parent vein (Fig. 1, *B*). If this closed sac is involved with structures destined for the mediastinum, it descends with them. If it is inextricably attached to those structures destined for the neck, its voyage is briefer and it manifests itself as a cervical lymphangioma. Occasionally a large closed sac may become involved with structures destined for both the neck and mediastinum. This type of development may manifest itself as a cervicomediastinal lymphangioma. The final position of the lymphangioma is determined by the destiny of the structures to which the displaced primitive lymphatic sac is adherent. Ten of the cases considered in this essay contained thin fluid which varied from clear to yellow or brown in color. This lends support to the postulation just given and indicates that these cysts neither regained connection with their parent vein nor entered into union with the stream of the lymphatic system.

Four of the cysts contained milky fluid which was clearly chyle. This state may be explained by recalling that after the primitive jugular sacs execute their connections with the veins and the remaining lymphatic system, they are transformed into lymph channels and lymph nodes. Thus, this developmental arrest would occur just prior to the transformation into lymph nodes. A similar example may be found in the adult anatomy in the form of the cisterna chyli. The precursor of this latter lymph receptacle is only partially transformed into lymph nodes and vessels.[20]

Two points are emphasized by the fact that the phrenic nerve was inseparably incorporated in our case of cystic lymphangioma and was in close relationship to cysts in this collected series. First, this intimate association is not surprising when it is recalled that branches of the third, fourth, and fifth cervical nerves pass through apertures in the primitive jugular sacs;[13, 20] second, as Goetch[7] has pointed out, "This sequestrated lymphatic tissue may retain the power of growth inherent in the process of endothelial sprouting and penetration characteristic of the development of the normal lymphatic system." Thus, lymphangiomas may tend to penetrate mediastinal structures, vessels, and nerves.

STRUCTURE

The shape of these tumors generally has been described as round or oval; ours was more saddle shaped. Touroff and Seley[28] described a cloacalike cystic structure filling the posterior mediastinum. Both unilocular and multilocular types have been noted. The size has varied from a few centimeters in diameter to one which occupied the entire lower hemithorax. Externally, the tumors have been shaggy and grayish white; the interior has usually presented a smooth, glistening surface with trabeculations.

Microscopically the fibrous wall has contained connective tissue and collagenous fibers. Smooth muscle often has been found in the trabeculations. This has appeared with sufficient frequency to suggest that it may be characteristic of these cysts. Perhaps a more extensive search would have revealed smooth muscle in a greater number of the collected cases. Lymphocytes, eosinophils, cholesterol crystals, and fatty acid crystals have been mentioned. In one case[27] a foreign body reaction was noted but in none of the others was a true inflammatory exudate found. On the internal surface a lining of flattened cells, usually termed endothelium, has been found. In one cyst no lining cells were found presumably due to an inflammatory reaction within the lumen from *B. coli* and enterococcus.[2]

Chemical analysis was performed on the thin, nonmilky type of contents from five cysts.[1, 2, 23] Protein, glucose, chloride, cholesterol, and lipids were found in varying quantities.

THERAPY AND RESULTS

All seem agreed that surgical excision is the procedure of choice. The main objectives of thoracotomy are to extirpate a space-occupying lesion and to establish a diagnosis.

It is not always possible to excise the cyst completely because of inseparable involvement of neighboring structures. Four excisions were incomplete for this reason[23, 26, 28] and one of these cysts recurred three years postoperatively.[23] Chemical sclerosis with Lipiodol[28] was utilized once. Gross and Hurwitt[8] report the use of iodine as a sclerosing agent in the treatment of the mediastinal extension of a cervical lymphangioma. Santy and associates[23] performed a biopsy and drained the cyst. They approached it through a cervical mediastinotomy as a mediastinal thyroid. Apparently this latter cyst still remains after repeated aspirations. A two stage excision was utilized on one occasion.[26]

In the 18 cases, there are listed one empyema, one wound infection, and two nerve paralyses as operative complications.

Most of the follow up periods are too brief to draw any conclusions as to recurrence rate. As noted above, there was one recurrence three years after an incomplete excision. However, the longest follow up period, ten years with no recurrence, was also in a case of incomplete excision.[25] It may be that the inflammation of the resulting empyema in this latter case obliterated the remaining small cysts.

CONCLUSIONS

1. Mediastinal lymphangiomas are probably due to developmental defects of the primitive jugular sacs and in this regard do not differ from cervical and cervicomediastinal lymphangiomas.
2. Whether the lymphangioma contains milky or nonmilky fluid probably depends on the stage of arrest of this developmental anomaly.
3. Mediastinal lymphangiomas are discovered later than cervical lymphangiomas because of the unyielding nature of the bony thorax.
4. There are no specific symptoms, sex, or age groups which are of aid in diagnosing these mediastinal cysts.
5. Certain patterns of anatomical location seem to exist, namely, in the lower anterior mediastinum and in the triangle between the superior vena cava, azygos vein, and esophagus.
6. Smooth muscle appears to be a frequent occupant of the cyst wall and trabeculae.
7. Lymphangiomas may be inseparably attached to mediastinal structures.
8. Exploratory thoracotomy is necessary to establish a diagnosis and to extirpate the cyst. Chemical sclerosis may be beneficial when incomplete excision is practiced.

SUMMARY

1. Seventeen cases of mediastinal lymphangiomas which were subjected to operative intervention have been collected from the literature.
2. An additional case (the eighteenth) is presented in detail.
3. The embryology and clinical manifestations of mediastinal lymphangioma have been presented and discussed.

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GIANT HEMOLYMPH NODE OF THE LEFT CHEST

Report of Case

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A CASE of giant hemolymph node of the chest is presented for the record, approximately seven years after its successful removal. The delay in making the report has been by intent. The patient's continuing excellent health, without evidence of recurrence or persistence of the disease, seems to be the added evidence to confirm the diagnosis.

By a most careful search of literature, I was able to find but 5 similar clinical cases. Symmers,[7] *in 1921, reported 2 cases which he described as* hemangio-lymphoma of the hemal nodes. In 1923, Lederer[2] described a similar case, and in 1950, Pemberton, Broders, and Maino[5] reported 2 cases. Leydig,[4] in 1857, described para-aortic lymph nodes in sheep and dogs not unlike accessory spleens. Gibbes,[1] in 1884, reported the finding of nodes, which contained circulating blood, in the region of the renal artery and vein. Robertson,[6] in 1890, published the gross and microscopic anatomy of these structures as found in sheep, bullocks, and human beings, and was the first to call them hemolymph glands. Warthin[8] made an exhaustive study, and, according to him, these nodes have an investing capsule of varying thickness, containing a little elastic tissue and varying amounts of smooth muscle and fibrous tissue from which irregular trabeculae project toward the hilum, dividing it into irregular lobules. Blood vessels enter the hilum and capsule, and communicating sinuses accompany the trabeculae. The subcapsular sinus contains blood and may extend around the entire periphery of the node. The lymphoid tissue between the sinuses varies greatly in amount, but otherwise resembles ordinary lymph nodes. Small lymphocytes are the chief cellular constituent and they may follow follicular arrangement. Usually, no arteriole is present in the follicle, in contrast to the spleen. *Polymorphonuclear leukocytes, large mononuclear cells, and* eosinophils are also present. Phagocytes containing erythrocytes are frequently misinterpreted as eosinophils.

Hemolymph nodes cannot be considered ordinary lymph nodes into which blood has extravasated, because they are found in conditions in which trauma and congestion can be excluded. Various functions have been attributed to hemolymph nodes, but phagocytosis, particularly of the erythrocytes, slightly greater than that of the spleen, is their most obvious function, according to Lewis.[3]

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Hemolymph nodes are found in the prevertebral area of the abdomen, the thorax, and the neck, particularly in the vicinity of the larger vessels. In man, hemolymph nodes vary usually between 2 mm. and 6 mm. in diameter. Giant forms of this structure, however, have been reported in man, but they are rare. Symmers,[7] in 1922, reported the cases of 2 patients with hemolymph nodes who came to autopsy. The first was a girl three and one-half years of age. The necropsy findings revealed an abdominal tumor 10 by 9 by 6 cm., with widespread metastasis, notably in the mediastinum and the subperiosteal tissue. His second case was that of a 28-year-old man who had died of cyanide poisoning and, at necropsy, a 6 by 5 by 4 cm. tumor was found overlying the left pelvic brim. The tumor had metastasized to the para-aortic region. Histologically, both tumors contained lymphoid cells associated with a large number of blood channels. In certain areas, there was distinct resemblance to the Malpigian bodies of the spleen.

In 1923, Lederer[2] found at necropsy, in a 14-year-old girl who had died of heart disease, large nodes in the region of the thymus gland. The largest node was 3 by 10 by 9 mm.

Pemberton, Broders, and Maino,[5] in 1950, reported 2 cases of giant hemolymph nodes. Their first patient was a 38-year-old woman seen in 1946. During the general examination, a mass was found in the left chest. At operation a tumor, 7 by 6 by 5 cm., was found adjacent to the vertebral column in the posterior part of the thorax and was removed. The second case, in 1949, was that of a woman, 40 years of age, with a complaint of a gradually enlarging tumor of the right anterior cervical region. This tumor was removed and histologically corresponded to the structure of the first case and both conformed to the description of hemolymph nodes as outlined by Warthin.[8]

CASE REPORT

The case I wish to present was referred to me on Nov. 6, 1947, by Dr. E. J. Murray of the Julius Marks Sanatorium. The patient was a white man, married, 24 years of age, and employed as a freight handler, which entailed heavy lifting. He had been in excellent health until three weeks previously. His illness was acute in onset with sharp pain in the anterior left chest, over the left scapula, and in the upper left abdomen. The pain was intensified when the patient breathed deeply or lay on his right side. A week later, shortness of breath appeared and increased progressively to a disabling degree. A cough productive of a small amount of clear sputum had been present for ten days. There was no history of chills, fever, or tuberculous contacts.

The patient, an intelligent, vigorous-appearing physical specimen, 6 feet tall, weighing 200 pounds, without history of serious illness, accident, or surgical experience, had been rejected by the Army in September, 1945, without being given a reason; however, his home physician, two years previously, had told him that he had a tumor in the left chest behind the heart. At the time of my examination, I saw an exceedingly well-nourished and developed young, white man of ruddy complexion in apparent good health except for moderately severe dyspnea. On examination, the whole left chest was dull to percussion with absent voice and breath sounds. The temperature was 99.4° F., the pulse 100, strong and regular, and the respirations were 26 to 30 per minute. The erythrocyte count was 5.15 million, with 96 per cent hemoglobin. The leukocytes numbered 11,300, with 82 polymorphonuclear, 18 lymphocytes, and 11 stab forms. The urine was normal both chemically and microscopically. Roentgenograms of the chest

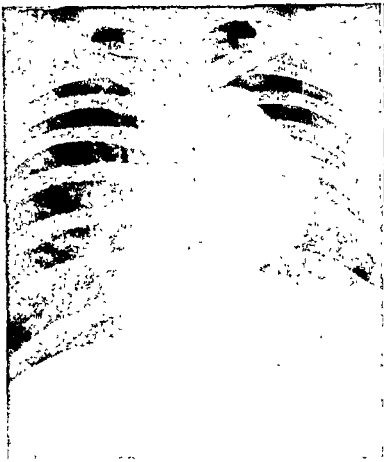

Fig. 1.

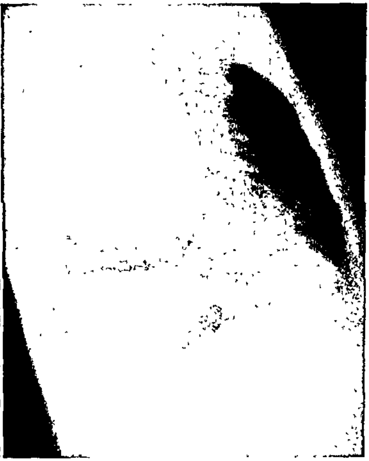

Fig. 2.

Fig. 1.—Posteroanterior view of chest which reveals a large tumor after aspiration of 9,536 c.c. of clear, straw-colored fluid from left chest.

Fig. 2.—Lateral view of chest which shows posterior position of the giant hemolymph node.

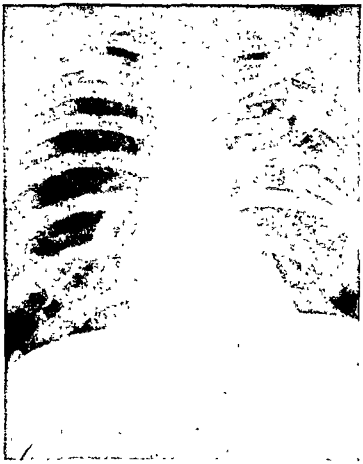

Fig. 3.—Postoperative view which shows complete removal of tumor.

were the most valuable single bit of evidence (Figs. 1 and 2). The initial films showed a dense homogeneous shadow involving the whole left cavity, with the heart displaced to the right. Within the succeeding four days, a total of 9,536 c.c. of clear, straw-colored fluid was aspirated. Smears, cultures, and guinea pig inoculations were done on the fluid and in time all proved to be negative. Sputum was never available for study. X-rays of the chest following aspiration revealed a large slightly irregular tumor, approximately 14 by 10 cm., projecting into the left mid-chest field.

On Nov. 12, 1947, a left thoracotomy incision was made through the resected bed of the sixth rib.

Approximately 300 c.c. of straw-colored fluid remained in the left chest cavity and was aspirated. Exploration then revealed a large, firm, slightly irregular rounded tumor slightly fixed in the posterior lateral gutter. The heart and great vessels appeared and felt normal and there was no extension or attachment to the mediastinum. The tumor extended posteriorly from about the fourth to the ninth ribs. The lower lobe of the lung was adherent to the anterior surface of the tumor by thin adhesions which were easily sectioned by sharp dissection. There was no invasion of the lung or bony structures, but the tumor was rather firmly attached to the posterior chest wall. A rather large area of parietal pleura was removed with the tumor. The postoperative course was uneventful and the patient was dismissed from the hospital on the fourteenth day (Fig. 3). The diagnosis of giant hemolymph node made this case of unique interest and as a consequence he has been closely followed. He resides in a near-by town, willingly returns for re-examination, and readily responds to letters of inquiry. In a recent communication, he stated that he was in excellent health. He maintains a weight of 200 pounds and is employed as a truck driver without restrictions. He is free from cough, sputum, chest pain, shortness of breath, palpitation or other disorder; in fact, he considers himself in unusually good health.

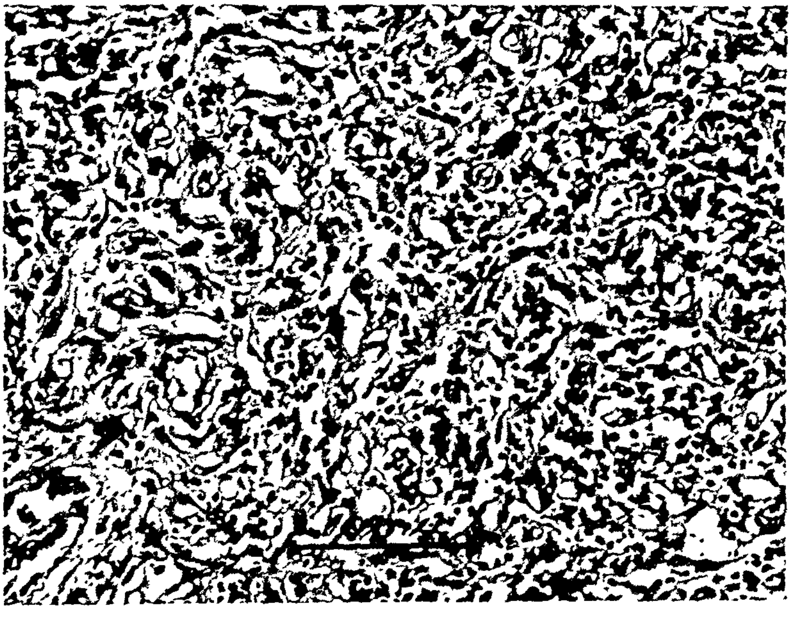

Fig. 4.—Giant hemolymph node. Small lymphocytes are the predominant cells and there are a large number of sinuses containing erythrocytes. No Sternberg-Reed cells were identified. A wide reticular framework surrounds the small vascular spaces and irregular, thick, fibrous septa divide the tumor into irregular lobules.

As might be expected in a condition of such rarity, the pathologic diagnosis was an added problem. I referred the histologic slides to Dr. A. C. Broders, then in the

Pathological Department of the Mayo Clinic, for his opinion. He kindly reviewed the slides and made an unequivocal diagnosis of giant hemolymph node and urged me to report the case because of its rarity.

The gross specimen was described as a tumor mass from the left chest cavity measuring 14 by 10 by 8 cm. It was enclosed by a fibrous capsule to which fibrofatty tags were adherent. On section, it was composed of moderately firm, pinkish-tan tissue divided by irregular fibrous septa.

Microscopically, the capsule was formed by a thin layer of collagenous connective tissue with fairly heavy vascularized fibrofatty tissue adherent to the outer surface. Fibrous septa, having something of the appearance of splenic septa, invade the substance of the tumor. The pulp between the septa is not that of ordinary splenic tissue. The basic pattern of the mass is lymphoid in type with evenly spaced, fairly uniform small follicles throughout. All the elements appear adult and fully developed. A fine reticular framework supports sinuses or vascular spaces closely packed with erythrocytes. The small lymphocyte is the predominant cell of the lymphoid tissue. Other cells present in small numbers are histiocytes, neutrophils, and occasional eosinophils. No Sternberg-Reed cells were identified (Fig. 4).

Diagnosis.—Consistent with giant hemolymph node according to Warthin's description.

Note.—The diagnosis of giant hemolymph node had been suggested by Dr. Albert C. Broders.

SUMMARY

A case of giant hemolymph node of the chest cavity is presented because of its rarity. The patient remains in good health, free from recurrence or other disease, seven and one-half years after operation.

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LOWER ACCESSORY LUNG COMMUNICATING WITH THE ESOPHAGUS AND ASSOCIATED WITH CONGENITAL DIAPHRAGMATIC HERNIA

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INTRODUCTION

THE anomaly referred to variously as lower accessory lung, supernumerary lung, or Rokitansky's lobe constitutes one of the rarest anomalies encountered in the chest. This tissue is usually located between the base of the left lung and the diaphragm, but several cases have been reported below the left leaf of the diaphragm. A unique feature is that there is no connection with the bronchial tree. A review of the available literature reveals no previously reported identical case and but few that closely resemble the deformity to be described in this communication.

Among the earliest reports published are those of Rektorzik,[8] and Rokitansky,[9] each of whom separately reported a case in 1861. Since that time, over 40 cases of lower accessory lobe have appeared in the literature. Extensive bibliographies have been given by several authors.[1-3, 6, 11, 12]

DeBakey[3] performed the first successful resection of this anomaly in 1950. That same year Leahy and MacCallum[7] reported the second. Although the majority of recorded cases of lower accessory lungs have been found at autopsy, the presence of this anomaly alone is not incompatible with life. Davies and Gunz[2] mention von Meyrburg's case (a child 5 years, 9 months of age), Rektorzik's case (a girl of 18 years) and Springer's case (a woman of 49 years). The presence of an associated congenital diaphragmatic hernia is not an infrequent finding in cases of lower aberrant or accessory lobe. Valle and White[12] reported an incidence of 29.7 per cent. It is believed that this case is the first recorded case of lower accessory lobe communicating with the esophagus, and associated with congenital diaphragmatic hernia, to be successfully treated surgically. A somewhat similar case was reported by Scheidegger[10] who described a case of a premature infant having an anomalous lobe of lung adherent to the pleural side of the left leaf of the diaphragm and connected to the stomach by a tract lined with gastric mucosa. His case was not associated with a diaphragmatic hernia, however.

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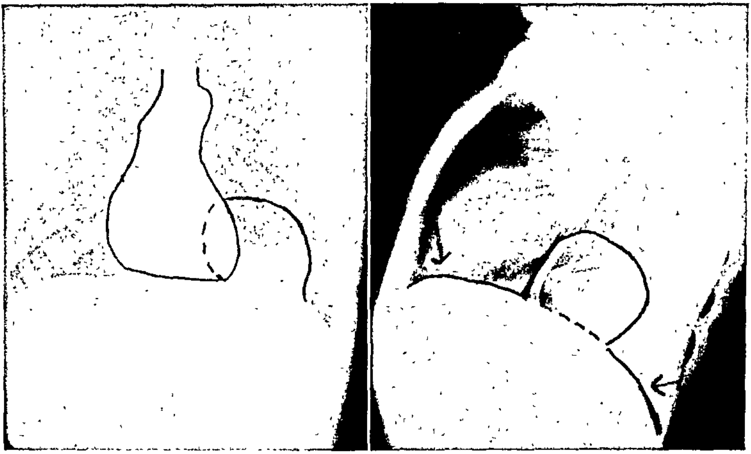

A. *B.*

Fig. 1.—*A*, Plain roentgenogram showing air-fluid level of stomach in lower left hemithorax. *B*, Lateral x-ray view within Levin tube passed into stomach demonstrating its intrathoracic position.

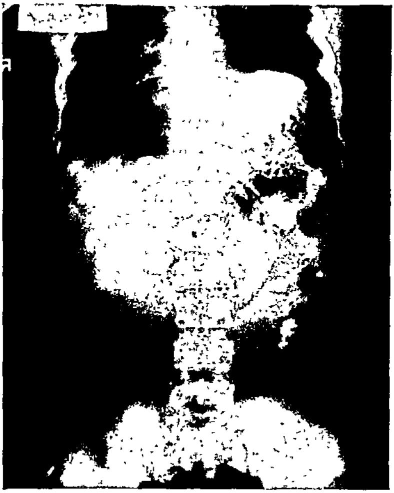

Fig. 2.—Barium study outlining intrathoracic position of portion of the stomach.

CASE REPORT

History.—M. D., a 13-year old Negro boy, was admitted to the John Gaston Hospital, Feb. 8, 1955, with the chief complaint of upper abdominal pain, which had recurred in mild attacks for the past year. Three weeks prior to admission, he had had an acute, severe attack while eating which was relieved by lying down. The last attack of epigastric cramping pain associated with nausea and vomiting had occurred nine days before admission. A routine tuberculosis survey roentgenogram had shown a translucent area in the left lower chest compatible with a hiatal hernia. The remainder of the history, including the systemic review, was essentially negative.

Physical examination revealed a well-developed, well-nourished Negro boy in no acute distress. The remainder of the physical findings were negative.

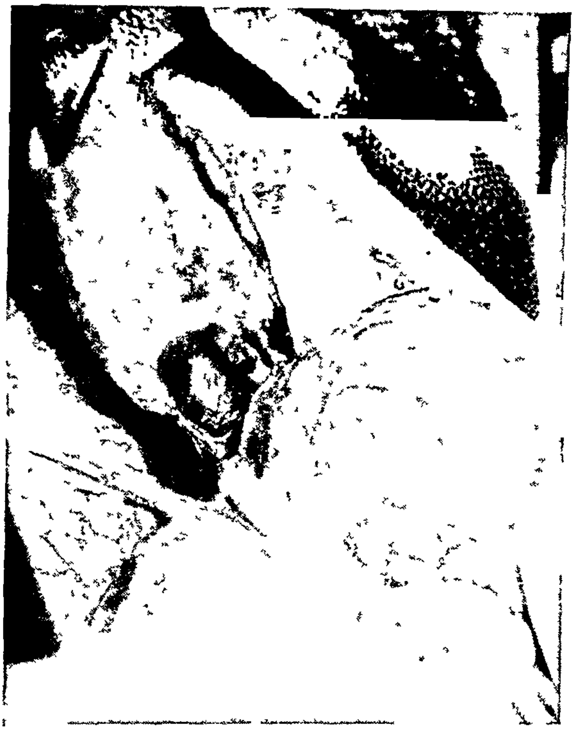

Fig. 3.—Close-up photograph at operation demonstrating diaphragmatic hernia and "accessory lobe" (being retracted upward with clamp). Note the pedicle of this lobe begins posteriorally in the sulcus between posterior thoracic wall and diaphragm adjacent to esophagus and aorta The large bronchus of this lobe entered the esophagus and the arterial blood supply was derived directly from adjacent aorta above the renal arteries.

Roentgen examination, which included a gastrointestinal series, confirmed the presence of the hiatal hernia with displacement of the upper one half of the stomach into the left chest (Fig. 1, *A* and *B*, and Fig. 2).

Laboratory studies were within normal limits.

Operation.—On Feb. 17, 1955, under endotracheal oxygen-ether cyclopropane anesthesia the left hemithorax was entered through the eighth intercostal space. On opening the pleural cavity, a diaphragmatic hernia was immediately visualized posteriorly. Contained within the hernial sac was the upper one half of the stomach and the greater portion of the spleen. Upon retracting the left lower lobe cephalad, a bluish-gray mass, roughly triangular in shape, was found lying in a posterolateral plane attached by a pedicle to the esophagus (Fig. 3). The mass was carefully dissected free and found to communicate with the esophagus by a patent passage just above the cardio-esophageal junction. The tract between the esophagus and the mass was doubly clamped, divided, and the esophageal opening closed with two inverting layers of No 3 0 silk The *arterial supply* originated from a branch of the abdominal aorta in the region of the renal vessels Venous drainage was into the portal system The hernial sac was opened widely, the stomach and spleen were returned to the abdomen, and repair of the diaphragmatic hernia was accomplished without difficulty

The *postoperative course* was essentially uneventful and the patient was discharged from the hospital on Feb 27, 1955

Pathology Report—The specimen received was consistent with lung tissue It measured 6 by 4 5 by 3 5 cm and showed a somewhat smooth anterior surface with a somewhat bulging posterior surface The lower portion of the specimen was composed of a very large vein and artery A large bronchus, measuring approximately 0 7 cm in diameter, which was connected to the esophagus, was present The bronchial wall was smooth and thin and shows no unusual anatomic picture The blood vessels were transversely seen in the specimen without showing any blood coagulation in the lumen

The final pathologic diagnosis based on histologic sections was that of accessory lobe of lung showing chronic interstitial inflammation

DISCUSSION

Etiology—No unanimity of opinion exists with regard to the origin of lower accessory lobe Of the numerous theories proposed, only the two which seem most plausible will be mentioned The theory of sequestration advocated by Cockayne and Gladstone[1] appears to be the most widely accepted They believe that a portion of the embryonic pulmonary tissue, after having become adherent to a neighboring part or organ from which it draws its blood supply, secondarily may be separated from the parent tissue and grow independently These adhesions of the lung bud to the septum transversum covering the liver, or to the wall of the pleuroperitoneal passage in typical left sided cases of accessory lung, may interfere with a normal retraction of the lung bud from the abdomen into the thorax and cause a persistence of the pleuroperitoneal opening This could explain the association of the accessory lungs with left sided congenital diaphragmatic hernia and also the attachment of the accessory lung to the lower esophagus and stomach, a finding in this case

The other prominent hypothesis, that of duplication advocated by Eppinger and Schauenstein,[4] suggests that these anomalies be interpreted as true third lungs rather than aberrant sequestrated lung fractions or lobes

Although an entirely convincing explanation of the derivation of lower accessory lungs remains to be found, an analysis of the reported cases in the literature reveals certain characteristics common to all cases

1 They consist of masses of lung tissue in various stages of differentiation occurring in the thorax, lower neck, or upper abdomen and not connected with the normal respiratory tract
2 Despite the fact that most cases are found at autopsy, this anomaly is not incompatible with life
3 In the human being, accessory lungs are much more common on the left side
4 The presence of an associated defect of the diaphragm predominantly left sided is a not an infrequent finding

5. Except for an occasional rare case, the blood supply of lower accessory lungs is not associated with the pulmonary vascular system, but is derived from the aorta.
6. Although lower accessory lungs are subject to the diseases which affect the normal lung, they are usually free of disease.

Our case presents several interesting points. The diaphragmatic hernia was detected by a routine tuberculosis survey x-ray film approximately three months prior to admission to the hospital and had produced no severe symptoms until approximately three weeks prior to admission. The lower accessory lung was not detected by the preoperative roentgenologic examination. Although a patent communication with the esophagus existed, the patient had experienced no severe respiratory infection. This is in marked contrast with the case reported by Gans and Potts.[5] Their patient, a 5-month-old infant, had experienced recurrent severe attacks of respiratory infection since birth. The anomalous lobe was located in the apex of the left chest and was not associated with diaphragmatic hernia.

SUMMARY

1. A case of a lower accessory lung communicating with the esophagus and associated with congenital diaphragmatic hernia is presented.

2. The possible origin of this anomaly is discussed.

3. Successful repair of the diaphragmatic hernia, with removal of the accessory lobe, was accomplished without incident.

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DIRECT EXPERIMENTAL APPROACH TO THE AORTIC VALVE

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INTRACARDIAC surgery under direct vision is the logical aim of those endeavoring to correct congenital or acquired lesions of the heart. Up to the present, blind procedures have, of necessity, provided the surgeon with his most widely applicable techniques, in both the clinical and research fields.[1-6] These limited maneuvers preclude the attainment of direct exposure of the structures involved and as such remain approximations to an ideal.

New developments in the fields of hypothermia,[7, 8] pump oxygenators,[9] and cross circulation techniques[10] have expanded the horizons of intracardiac maneuvers under direct visualization, making it possible to stop the inflow and outflow tracts of the heart for varying periods of time and to attack most intracardiac problems directly under circumstances which promise low rates of morbidity and mortality.

The region of the aortic valve stands out conspicuously as a difficult one to approach even with the newer techniques. Aside from minor obstacles of a technical nature, such as high intraluminal pressures and/or friable mural tissues, free entry into this area exposes the coronary ostia and leads invariably to coronary air embolism, myocardial anoxia, ventricular fibrillation, and death. Although some observers have demonstrated the reversibility of these changes when coronary air embolism and/or ventricular fibrillation[11, 12] are produced as isolated experimental situations, it is logical to assume that if they occurred concurrently with manipulations at the aortic valve area the associated mortality would rise significantly.

A number of techniques have therefore been devised to approach the aortic valve. Those meeting with success in the experimental animal and in the human being are invariably blind procedures involving:

1. Introduction of ingeniously designed instruments through the left ventricle with the purpose of fracturing or dilating the aortic valve from below.
2. Application of various grafts of pericardium, vessels, and plastic prosthesis to the base of the aorta, and introduction of the manipulating finger or instrument through these tunnels to obtain the desired intracardiac valvular effect.

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Our first approach to the problem was to prevent air embolism of the coronaries by cessation of cardiac contractile activity whether by electric shock, with ensuing fibrillation, or by perfusion of the coronary system with potassium chloride with ensuing cardiac arrest in asystole. The proximal aorta would then be opened, the desired defect produced, and the aortic incision closed under water to prevent entrance of air after normal contractile activity was re-established.

These efforts failed invariably, in our hands, because of the difficulties encountered in restoring persistent normal cardiac rythym for significant periods of time after the aorta was closed.

It has been the purpose of this investigation to develop a suitable approach to the aortic valve under direct vision and concomitantly eliminate the complication or air embolism to the coronary arteries. The result of these experiments represents a phase in this project. Our final goal will be directed toward developing a method by which the condition just mentioned would be satisfied and the period of time available for direct valvular surgery be adequate.

METHOD

Adult mongrel dogs weighing from 7 to 10 kilograms were employed. The animals were anesthetized by the administration of 30 mg. Nembutal per kilogram of body weight. Some were cooled to 27° to 28° C. rectal temperature with a refrigeration unit equipped with cooling blankets. An intratracheal tube was inserted and artificial assistance to respiration carried out by an automatic respirator.

The thorax was entered through an incision in the right fourth intercostal space. The superior and inferior venae cavae were then isolated over umbilical tapes preparatory to future occlusion. The azygos vein was ligated in continuity, thus securing control over cardiac inflow.

The pericardium was incised longitudinally, parallel and 1 cm. anterior to the right phrenic nerve, and its borders were sutured to the thoracic wall, lifting the heart upward to provide better exposure. The tip of the right auricle was grasped with a hemostat and ligated so it would be pulled away from the aorta, exposing the proximal aortic surface. Dissection of the anterior aortic fat pad was then carried out and both posterior and anterior fat pads were grasped with hemostats for traction, in order to lift the vessel into closer view.

At this point dissection at the aortic origin was abandoned and, after mild rotation of the heart, a purse-string suture was placed in an avascular area of the apex of the left ventricle.

A stab wound was made in the center of this area, entering the ventricular cavity and through this wound a No. 20 polyethylene catheter was introduced. The latter had been previously connected to a saline reservoir. Care was taken that all tubing up to the catheter tip was filled with fluid.

The purse-string suture was tied snugly about the catheter securing it to the ventricular wall. Preparations were then made to enter the aorta and, with all instruments in readiness, the superior and inferior venae cavae were occluded by tightening the umbilical tapes about them.

Exerting upward traction on the fat pads, as described previously, the aortic origin and its sinus of Valsalva were brought into prominence so that an incision about 1.5 to 2.0 cm. in length could be made along the long axis on its anterior wall.

Upon entering the lumen of the artery, a steady flow of saline solution was allowed to enter the left ventricle via the polyethylene catheter, and in a few seconds this washed the blood from the ventricular cavity through the aortic incision. The structures at the aortic valve were then clearly visualized. Exposure of the valve was enhanced by placing a small retractor at the proximal end of the aortic incision, carefully avoiding tearing the vessel wall.

A suture of No. 4-0 silk was then placed to include two adjacent cusps near their mural attachments. During suture, the saline stream could be controlled so that it merely covered the intimal surfaces and the coronary ostia, and did not obscure the operative field.

Upon tying the aortic valvular suture, the stream was again increased and suture of the aortic incision was begun. Simultaneously with or just previous to the conclusion of aortic suture, the saline stream was discontinued to prevent overdistention of the left ventricle. The cardiac inflow tract was also opened slowly, releasing the constriction on the superior vena cava first and that on the inferior vena cava a few minutes later. By the time aortic closure was completed, the heart had assumed strong regular contractions and the ventricular irrigating catheter was withdrawn followed by the placing of a suture to close the small ventricular wound.

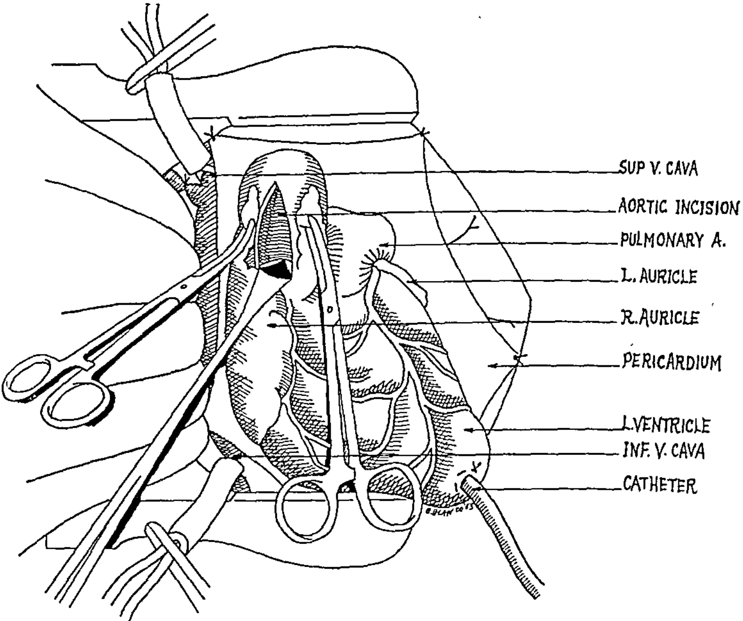

Fig. 1.

All pericardial sutures were divided, the incisions were inspected for evidences of bleeding, and the heart was allowed to resume its normal position. The pericardium was sutured loosely over the heart in a few instances.

Both pleural cavities were made dry of saline solution and blood by suction, and the chest was closed in layers leaving a rubber intercostal catheter in the right costophrenic sulcus to which suction was applied.

The hypothermic animals were then rewarmed; a process which usually took one and one-half to two hours. By the end of this period, spontaneous respirations had reappeared, making the use of a respirator unnecessary. Four hundred thousand units of penicillin were injected through the intercostal catheter and it was withdrawn. The animal was returned to his cage.

Electrocardiographic tracings were obtained before and after surgery.

DISCUSSION

Twenty dogs were operated upon by this method; 11 were hypothermic, 9 were normothermic. During the period of inflow tract occlusion, intra-aortic maneuvers could be carried out for 2 to 4 minutes, allowing ample time for commissural suture with the production of varying degrees of stenosis. Exposure of the valve leaflets was adequate after a short (15 to 20 seconds) initial period to allow clearing of the left ventricle of its contained blood by the irrigating saline solution.

Twelve dogs exhibited ventricular fibrillation which usually occurred at the termination of aortic closure. The onset of this arrythmia coincided in many cases with release of the cardiac inflow tract. In these dogs, ventricular contractions, which had been getting progressively weaker, would become almost imperceptible. It was observed that if the stream of saline solution was not cut off just previous to this moment, or if both superior and inferior venae cavae were released simultaneously, marked distention of the right chambers would occur with the appearance of arrythmia. Fibrillation was treated with the usual measures of massage, intracardiac Adrenalin, and electric shock.

Ten dogs succumbed within forty-eight hours of the procedure and are considered operative deaths. Of these, only 2 died during the operation, both from irreversible ventricular fibrillation. One of these is the only case in which air embolism of the coronary circulation occurred, and in this dog the ventricular catheter became dislodged so that the coronary ostia were exposed to air for at least 2 to 3 minutes with resultant massive air embolism.

The remaining 8 early deaths can be attributed almost exclusively to technical errors. Insecure aortic closure resulted in cardiac tamponade (1 case), and hemorrhagic shock (2 cases).

One of the early cases was lost at the table (previously mentioned) because of persistent ventricular fibrillation at the time both inflow tracts were released.

Although measures to stop ventricular fibrillation appeared to be successful in many cases, recurrence of fibrillation must explain some of the remaining deaths. Again, undue prolongation of intra-aortic maneuvers, particularly in normothermic animals, must have caused irreversible cerebral damage and contributed to the high mortality.

SUMMARY

1. A direct surgical experimental approach to the aortic valve is presented.
2. Twenty dogs were operated upon by this method and the results are discussed.

3 Air embolism to the coronary vessels is eliminated by this technique

4 A 50 per cent mortality within forty eight hours is reported, ascribed in large measure to technical errors during the operative procedure

5 The surviving animals will be studied with regard to physiologic and pathologic changes resulting from aortic valve suture

6 The short duration of the available operating time for intra aortic maneuver and the high incidence of ventricular fibrillation limit the possibilities of any clinical application at this time Further experiments are in progress in an attempt to correct these deficiencies

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EXPERIMENTAL METHODS FOR PRODUCING A COLLATERAL CIRCULATION TO THE HEART DIRECTLY FROM THE LEFT VENTRICLE

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CURRENT operative procedures designed to re-establish adequate myocardial circulation in human and experimental occlusion of the coronary arteries can be grouped under the following categories: (1) application of an inflammatory agent like powdered talc or asbestos to the abraded pericardium to create a vascularized granuloma with or without partial occlusion of the coronary sinus,[1] (2) arterialization of the coronary sinus by placing a free vein graft between the aorta and coronary sinus or by direct anastomosis,[2] (3) the grafting of vascularized tissue to the myocardium (omentum,[3] mediastinal fat,[4, 5] skeletal muscle,[5] and other tissues), and (4) the implantation of a third artery (internal mammary) into the left ventricle.[6] In these operations, the improvement of the coronary circulation is achieved: (1) by extracardiac vascularization, and (2) by stimulating the development of intercoronary arterial anastomoses.[7, 8]

The existence of a large and constant supply of arterial blood in the left ventricle made it logical to infer that the circulation to ischemic heart muscle could be materially improved if a practical method were devised for channeling blood in functional amounts from the ventricular chamber to the surrounding myocardium. Accordingly, experiments were performed on dogs with intramyocardial arterial and polyethylene tube grafts.

The purpose of this paper is to describe three surgical methods for applying the foregoing principle and to compare their efficiency by subjecting the grafted animals, together with suitable controls, to test ligation of the anterior descending coronary artery.

METHODS

1. *U-Shaped Arterial Grafts* (21 dogs).—Open pericardiotomies through an incision in the fifth interspace, with intratracheal Nembutal-oxygen anesthesia, were performed under standard operating room conditions upon dogs of mixed breed weighing from 12 to 23 kg. A segment 4 to 6 cm. in length was excised from the left carotid artery through a separate incision in the neck.

This study was supported by a grant from the Los Angeles County Heart Association.

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Approximately ten openings, of 1 to 3 mm. each, were made by removing portions of the arterial wall with fine, sharp scissors. The prepared free carotid arterial graft was threaded through the left ventricular wall in an area supplied by the anterior descending coronary artery to form a U-shaped perforated graft with the loop lying free in the heart cavity and the ends sutured loosely to the epicardium. Thus, new channels were created for blood to flow directly from the ventricular chamber into the heart muscle via the lumen of the arterial graft as well as through the spaces between the graft and myocardium (Fig. 1). In addition, the proliferating endothelium, by way of the perforations, was afforded an opportunity of fostering anastomotic channels with surrounding myocardium.

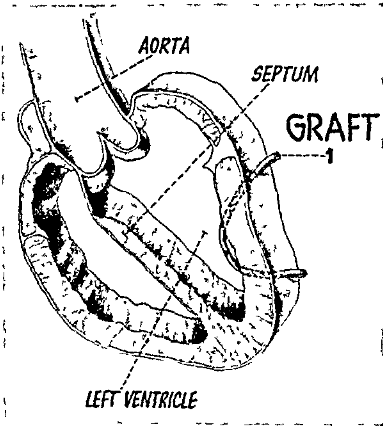

Fig 1.—Free carotid artery graft

In 2 groups of these prepared animals, after three weeks and two months, respectively, a second pericardiotomy was performed to test the protective effect of the arterial graft against acute myocardial ischemia. The myocardial circulation was challenged by ligating the anterior descending coronary artery immediately distal to the circumflex branch. No special effort was made to include the septal branch in the ligation. The pericardial incision was made small in order not to disturb newly formed adhesions. The pericardium was sutured loosely and the chest wall closed in layers without drainage. Twenty-four hours or five days later, the surviving dogs were anesthetized and sacrificed by opening the thoracotomy wound and removing the beating heart. Procaine penicillin, 500,000 units intramuscularly, was given for two to three days after each surgical procedure. In some instances, India ink, in amounts from 1 to 2 c.c., was injected under low pressure through the pericardial end of the arterial graft. The removed heart and pericardium were fixed in 10 per cent formalin.

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Measurements are in millimeters and *A B* and *C D* represent the width and length of the infarcts, and *W Z* and *X Y* the width and length of the ventricles (Fig 2)

Beginning at the apex, the heart was cut with an electric slicer into sections 2 to 3 mm thick, at right angles to the anterior descending coronary All of the slices containing the infarct were then replaced in their proper sequence one against the other, and the distance in millimeters between the first and last slices was taken to represent the length of the infarct (*A B* in Fig 2) The longest transverse measurement of the infarct area in any of the slices was arbitrarily taken as the width of the entire infarct (*C D* in Fig 2)

The third dimension (N) was graded 1 to 5 as follows (1) Absence of gross infarction (2) Patchy intramural infarction (3) Subendocardial necrosis with or without septal involvement (4) Subepicardial involvement in addition to N 2 and 3 (5) Necrosis through and through the ventricular wall

B *Pericardial Reaction*—This was graded 1 to 4+, as follows 1+, few filmy adhesions, 2+, extensive, filmy adhesions, 3+, thick adhesions, and 4+, thick extensive adhesions which when separated by blunt or sharp dissection while the heart was beating left oozing epicardial and pericardial surfaces

The slices were studied grossly for location and patency of the graft and size and distribution of the infarcts Suitable microscopic sections were prepared from these slices

RESULTS

U Shaped Grafts—(Tables I and II) The 3 groups of animals employed showed a greater degree of protection against ligation than any of the other groups The percentage of survivors was greater and infarct size smaller

TABLE I RESULTS IN CONTROLS AND U GRAFTS IN LEFT VENTRICLE

GROUP	NUMBER OF DOGS	PER CENT SURVIVING AFTER CHALLENGE	SIZE OF INFARCT (MEAN IR)	PERICARDIAL ADHESIONS
Normal controls	18	39	59 8	1+ 2+
Chromic phosphate controls				
a P^{32}	7	57	54 7	0 1+
b No P^{32}	7	57	57 8	0 1+
U graft 2 months	7	100	51 0	3+ 4+
U graft 3 weeks	7	85		3+ 4+
Multiple U grafts 2 months	7	100	34 7	2+-4+

TABLE II INFARCT SIZE IN MULTIPLE U GRAFTS IN 7 DOGS

NUMBER OF DOG	A B*	C D*	W Z*	X Y*	IR*	N NUMBER*
18 MG 5	4 5	4 5	8 5	7 0	40 7	4
26 MG 6	3 0	4 0	7 0	6 0	28 5	2
25 MG 8	6 5	4 5	8 5	5 5	60 1	3
98 MG 10	5 0	4 0	7 0	6 5	44 0	2
100 MG 12	4 8	4 5	7 6	7 4	37 1	3
76 MG 14	3 5	4 0	9 0	7 0	22 2	2
24 MG 15	2 5	1 5	6 5	5 5	10 4	2

*See text for definition of symbols and compilation of IR

(Figs. 3*A* and 3*B*). Ballooning and cyanosis immediately after ligation were inconspicuous.[7] In each of the groups, vascularized pericardial adhesions were well developed especially about the pericardial ends of the grafts. When these adhesions were cut while the heart was beating, there was sustained oozing of bright red blood from the muscular side around the graft as well as from the pericardial side. In the 2-month groups, denser, less vascular adhesions were consistently found over the distal anterior descending coronary. Gross examination demonstrated patency of all grafts. The patency was not always uniform in a given graft; as a rule, the portion which projected into the ventricle re-

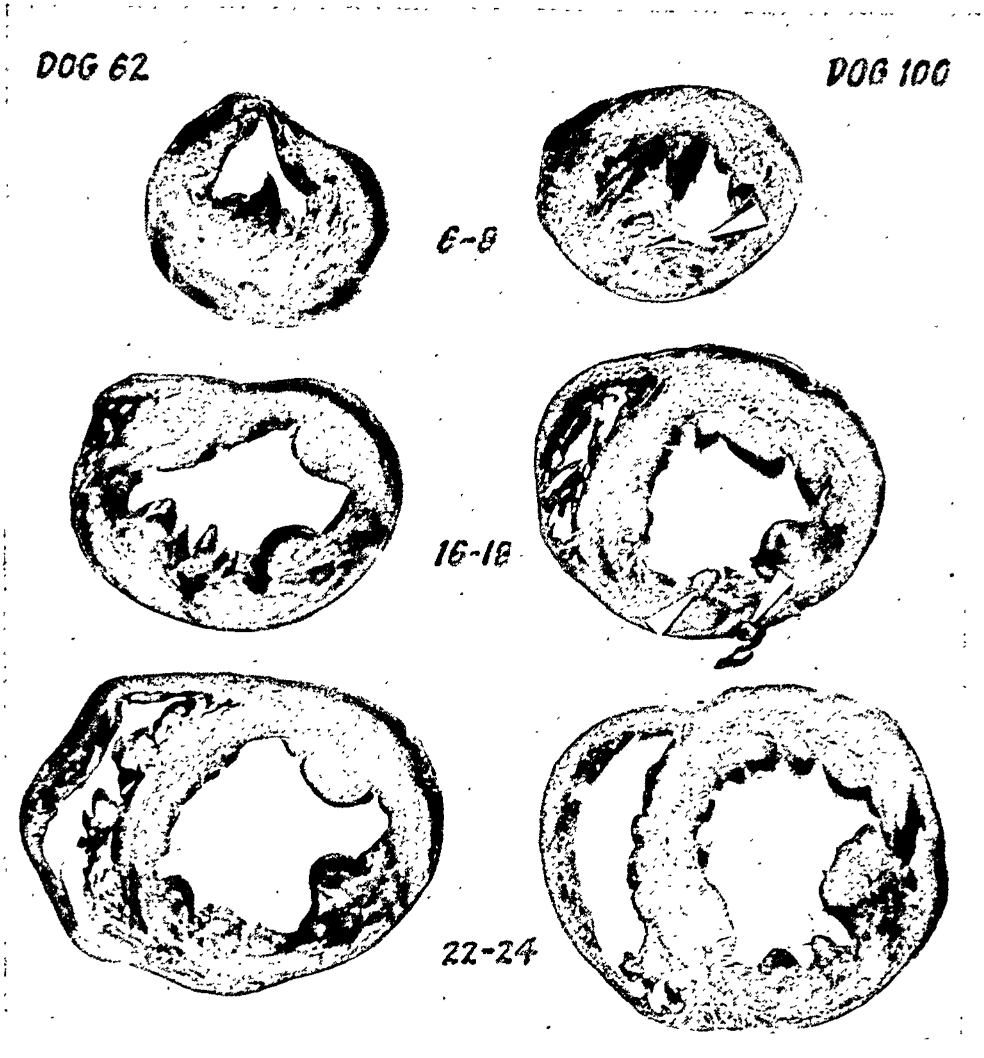

Fig. 3*A*.—Comparison of infarcts in control Dog No. 62 with 5-day infarct; and Dog No. 100 MG 12, multiple (2) U-graft with 5-day infarct.

Control IR = 59.1 N = 4
U-graft IR = 37.1 N = 3

Comparable slices at 6 to 8 mm., 16 to 18 mm. and 22 to 24 mm. from apex. Note the smaller size and patchy nature of the U-graft infarct compared to the control. White markers point to the U-grafts.

tained its patency more satisfactorily than the intramural segment. Microscopically, there was only minimal necrosis even after two months, despite the fact that the grafts had remained in acutely ischemic areas up to five days after challenge. Frequently there was free blood in the lumen. Hematoxylin-eosin stains showed pronounced intimal proliferation but also, in some but not all, varying degrees of degeneration of the arterial wall (Fig. 4). In the hearts injected with India ink, many newly formed communicating vessels were demonstrated within the ischemic area (Fig. 5).

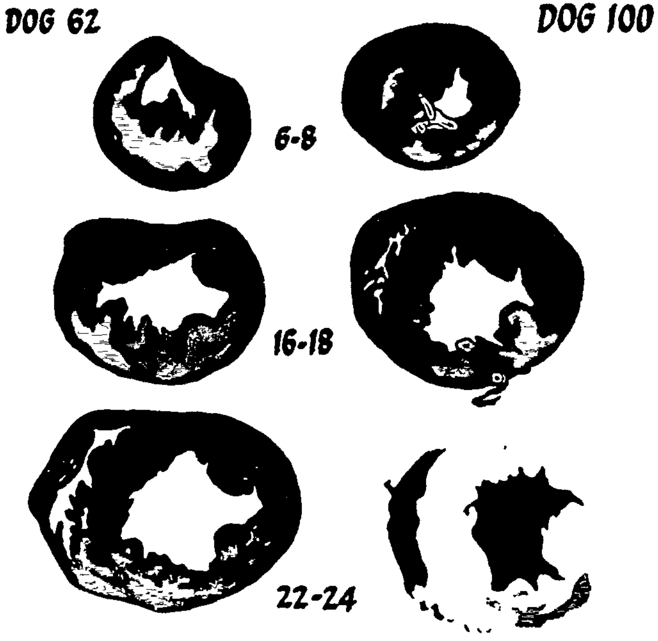

Fig 3*B*—Camera Lucida tracings of infarcts in Fig 3*A* Note smaller size infarct in Dog No 100 MG 12 prepared with U-grafts

Straight Arterial Graft.—Four dogs comprised this group At the time of challenge, three weeks after implantation, there was thrombosis in each instance in the portion of the graft which projected into the cavity of the ventricle Yet, 3 of the 4 (75 per cent) survived the ligation and in these the grafts were well tolerated by the animals without loss of weight. Pericardial adhesions were 4+ in 3 dogs, and 1+ in 1. The presence of thrombosis in the intracavitary projection of the graft and the microscopic findings indicated that at least some of the blood reached the myocardium via the myocardial space surrounding the graft.

Polyethylene Tubing.—Ten dogs comprised this group. Five dogs died before the end of the preliminary period of three weeks; 2 from embolism, 1 from pneumonia, 1 from hepatitis, and 1 from an error in ligation technique. The dogs tolerated the polyethylene graft poorly and all the grafts were thrombosed. Only 3 of the 10 animals could be challenged by ligation and but *one* of these survived the challenge.

Normal Controls.—In the 18 dogs which were used, death occurred within twenty-four hours in 11, or 61.1 per cent, and all but a few died before the end of the first hour after ligation. The behavior of these animals after the coronary artery was tied was very different from those prepared with U-shaped grafts. Ballooning and cyanosis were conspicuous and the rapid onset of myocardial ischemia was further suggested by the early appearance of ventricular fibrillation which often developed within minutes. The mortality was approximately two and one-half times as great and the size of infarcts was greater, only a mild pericardial reaction developed and, microscopically, there was evidence that myocardial repair was delayed. Estimation of the third dimension of infarcts showed many with ratings of N 4 and 5.

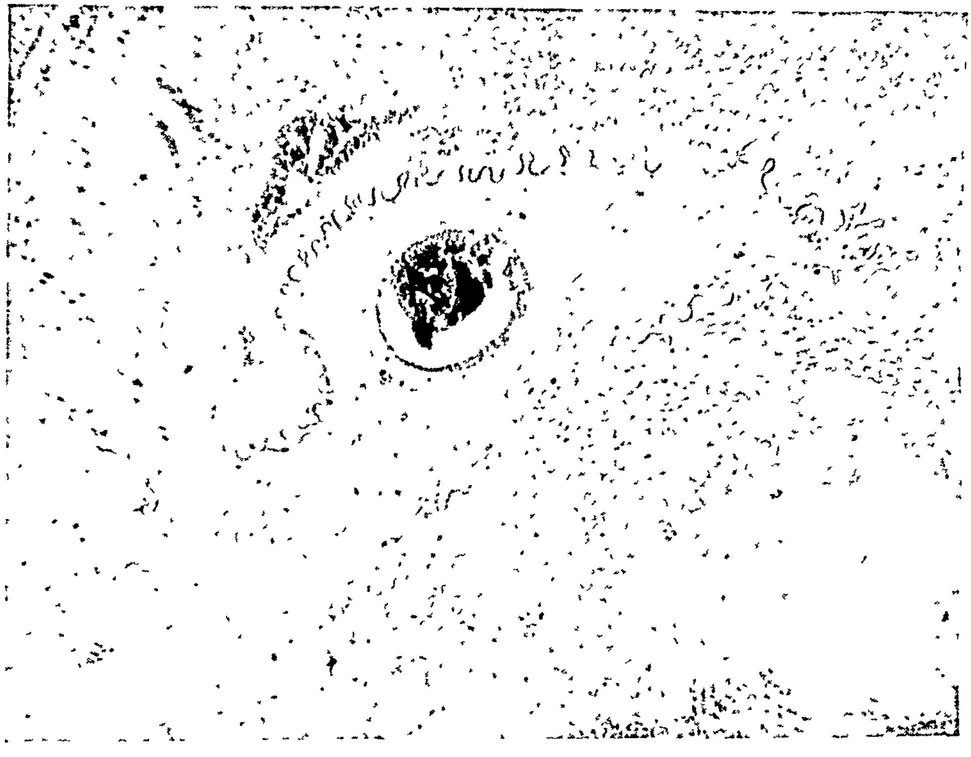

Fig. 4.—Dog No. 25 MG 8: U-graft in place for two months and in infarcted area five days. Note the necrosis of arterial wall, intimal thickening, narrowing, and thrombosis. Adjacent large vascular space with free blood is also present. (Microphotograph ×50.)

Chromic Phosphate Controls (Sham Operations).—Ballooning and cyanosis were as pronounced and as frequent after ligation as in the normal control group and the IR was not significantly different. On the other hand, the proportion of survivors indicated that the intrapericardial instillation of chromic phosphate provided some degree of protection against challenging ligation (Table I). The results in the group which received the radioactive compound were essentially the same as those treated with the nonradioactive salt, and the tests of benefit by survival and infarct size indicated that these compounds were of no value in augmenting the coronary circulation.

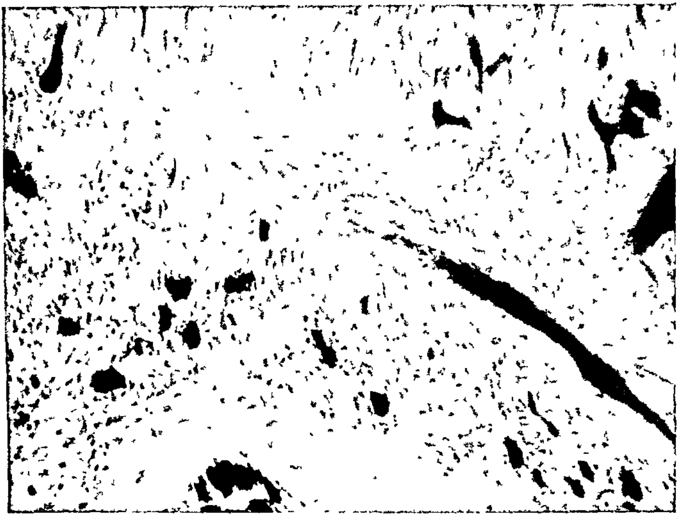

Fig. 5.—Dog No. 98 MG 10; U-graft in place two months and in infarcted area five days. India ink injection. Note that the graft is surrounded by numerous small vessels filled with India ink. (Microphotograph ×120.)

DISCUSSION

In an effort to improve the circulation of the myocardium in artificially induced ischemia, arterial grafts, both single and multiple, were inserted into the wall of the heart in such a way as to provide direct and immediate access of ventricular blood to the heart muscle. In the series of U-grafts animals tested against suitable controls, such a grafting operation did provide a certain amount of protection against the effects of ligation of a major coronary artery, as measured by infarct size and survival time. The difference between survival in the 18 normal control and the 21 U-graft dogs (Table I) was analyzed and these results were found to be statistically significant.* These favorable effects on survival statistics are further enhanced by the results in the chromic phosphate controls, which as sham operations increased survival only insignificantly.

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*These results were analyzed statistically according to a method of chi square described by Fisher and Yates[9] and applied to tests of significance for 2 × 2 contingency tables with one degree of freedom. This method is recommended where the smallest expectation of any class is less than 100. By this method chi square was equal to 11.9 and the difference therefore between the controls and experimentals was significant.

An unfavorable factor was the development of occlusive narrowing and progressive loss of mobility of the grafted artery. but it is notable, in some instances, that blood continued to reach the myocardium from the ventricle through the thickened grafts and especially via spaces and new vessels around the occluded graft. Gregg and Eckstein[10] have stated in reference to vessels embedded in myocardium that "when blood from the aorta flows through an embedded (arterial) segment a systolic flow may occur." It is reasonable to assume that a similar systolic flow should occur from the left ventricle through the thick-walled U-grafts. Beck and Leighninger[11] have accentuated the fact that relatively small quantities of blood (1 to 5 c.c. per minute) are sufficient to inhibit fatal trigger mechanisms arising in ischemic muscle. It is believed that the U-grafts should have been able to supply such needed amounts to the myocardium.

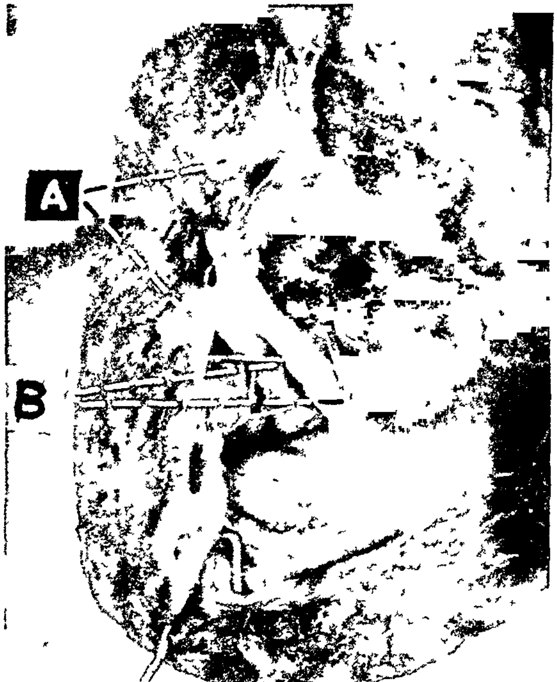

Fig 6.—Dog. No 82 MG 16 sacrificed three and one-half months after preparation with two U-grafts. The metal probes are placed inside the patent lumens of the grafts Note thickening of the walls of the grafts, and patency of both the intramural portions, *A*, and the intraventricular loops, *B*.

The ultimate fate of these free arterial grafts is not yet known and longer term experiments are already under way (Fig. 6). An additional group of 7 dogs with 3 parallel U-grafts has already survived in good general condition for four to six months, indicating that these grafts are well tolerated for such longer periods. The testing of the survival of these free grafts by the present experiments is the severest kind of test because they are placed in an acutely infarcted area, and the microscopic observations indicate that the artery survives better in the healthy muscle than in the infarcted area

It is premature to state that the principle of direct transfer of arterial blood from ventricle to myocardium can be applied to the surgical treatment of human coronary occlusion More studies are needed to investigate the survival of grafts in chronic experiments, to explain the mechanism of protection when grafts become occluded and to ascertain the value of grafting in hearts which have survived the effects of acute or chronic coronary occlusion

SUMMARY

1 A new principle involving free arterial grafting for revascularizing the dog's heart directly from the left ventricle was explored and 3 operative techniques described U shaped and straight carotid grafts were implanted in the left ventricle in such a way as to channel arterial blood directly from the heart chamber to the myocardium

2 Dogs with arterial grafts were challenged by ligation of the anterior descending coronary artery Protection in the grafted dogs was tested by survival and infarct size, and a comparison made with the results in controls A practical method for estimating infarct size was described Significant protection against ligation was observed, notably in the animals which received the U grafts Twenty of 21 dogs, or 95 per cent, survived ligation after implantation of single or multiple U grafts as compared with 39 per cent in controls

3 Polyethylene tubes engrafted into the heart and colloidal chromic phosphate, radioactive and nonradioactive, instilled into the pericardial sac gave disappointing results

4 A degree of protection was achieved even when thrombosis developed in the arterial graft, suggesting that blood may have reached the myocardium via channels around or connected to the artery

5 A brief report was made on promising long term experiments now in progress

6 The application of the grafting principle to human surgery was discussed

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Erratum

In the article by DR. CHARLES C. MACKLIN, entitled "Induction of Bronchial Cancer by Local Massing of Carcinogen Concentrate in Outdrifting Mucus," which appeared in the February issue of the JOURNAL, pages 238-244, the last two sentences under the heading 'Trachea" on page 243 should read as follows:

"If the cancer-producing action were primarily on the first part of the air tract encountered by the entering carcinogens we would expect to find the trachea more heavily involved than the great bronchi. By the time the mucus gets to the trachea it is doubtless depleted in carcinogens."

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Original Communications

THE SURGICAL TREATMENT OF AORTIC STENOSIS

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DEFINITION

THE term aortic stenosis refers to a significant narrowing of the orifice of the aortic valve. While it may be related to the presence of a constricted valve annulus of congenital or acquired origin, it usually is the result of intrinsic stiffening of the leaflets or cross fusion of their free margins (commissural obliteration).

HISTORICAL OBSERVATIONS

Riverius,[1] in 1647, apparently was the first to describe the pathologic changes of what undoubtedly was calcareous aortic stenosis, and to correlate them with the clinical features of a weak pulse and diminished arterial pulsations. Rayger,[2] Cowper,[3] Vieussens,[4] and Morgagni[5] have contributed early to the understanding of this entity.

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N J, April 24-26, 1955

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All of the catheterization and other physiologic data were supplied by Dr. Harry Goldberg and the Brith Sholom Cardiopulmonary Laboratory at Hahnemann Medical College and Hospital, Philadelphia, Pa

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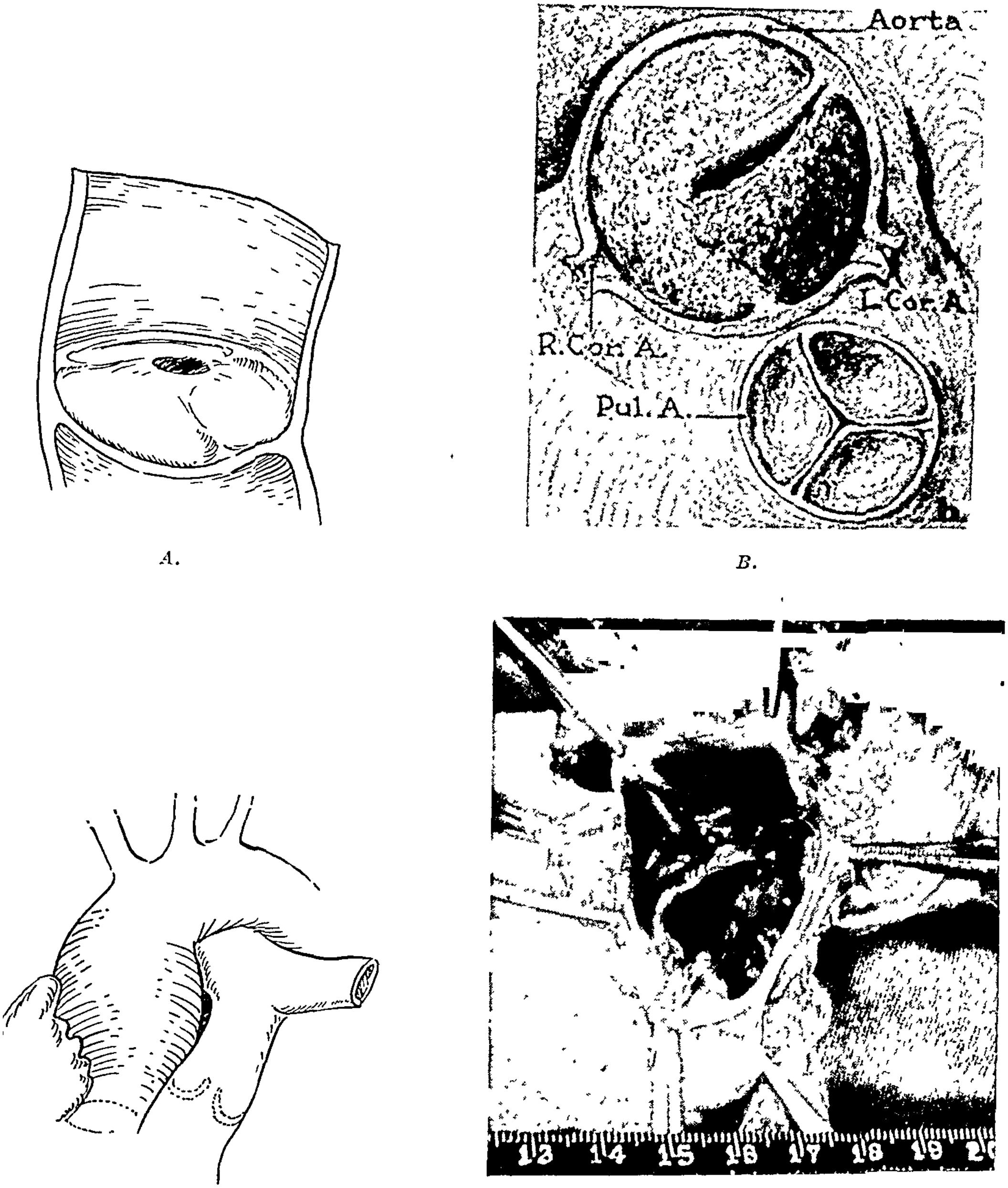
ETIOLOGY

It is believed that clinical aortic stenosis may be caused by congenital deformation, by arteriosclerotic changes in the aortic wall and valve leaflets, by the



A. B.

C. D.

Fig. 1.—*A*, Caplike congenital fusion of aortic valve cusps. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

B, Asymmetric congenital fusion of aortic valve cusps with only one commissure open. (From Taussig, Congenital Malformations of the Heart, courtesy Commonwealth Fund.)

C, Poststenotic dilatation of ascending aorta in congenital aortic stenosis. This also may be observed in certain patients with long standing acquired stenosis. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

D, Congenital fusion of two aortic leaflets producing a bicuspid valve. Note rudimentary raphe dividing the larger cusp. This patient exhibited aortic regurgitation as well as stenosis.

ulcerative and healing phases of bacterial endocarditis superimposed upon a previously abnormal valve, and, most commonly, by the effects of rheumatic valvulitis, alone.

Congenital Aortic Stenosis.—It is clearly evident that aortic stenosis may be of congenital origin. In these cases, there may be a generalized hypoplasia of the first portion of the aorta and the valve, often associated with a subvalvular stenosis. In other instances, the valve may present a "cap-like" or "megaphone-like" fusion of the cusps, forming a funnel-shaped diaphragm with a small centrally or eccentrically located orifice. The latter may represent an incomplete obliteration of one commissure (or two). In congenital cases, a post-stenotic dilatation of the ascending aorta is usual (Fig. 1, *A*, *B*, *C*). This phenomenon also may be observed sometimes in cases of long-standing advanced acquired aortic stenosis.

Another type of congenital aortic stenosis is that which is associated with a bicuspid aortic valve. However, not all bicuspid aortic valves are stenotic; some are associated with regurgitation. In most of these valves, two of the commissures will be normal but one (usually the anterior) will be found to be underdeveloped or incomplete. It may be represented by a rudimentary raphe dividing the larger of the cusps (Fig. 1, *D*). This type of valve frequently is found in association with coarctation of the aorta. Indeed, Reifenstein and his associates[6] have reported that 42.7 per cent of their series of 104 patients with coarctation also had a bicuspid aortic valve.

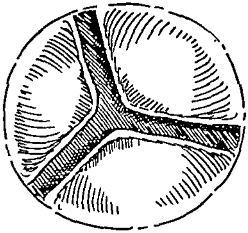


Fig. 2.—Arteriosclerotic aortic stenosis does not produce obliteration of the commissures. Instead the leaflets may become stiffened and springy but preserve their natural anatomical contours. (From Bailey: Surgery of the Heart, courtesy Lea & Febiger.)

Arteriosclerotic Aortic Stenosis.—Whereas formerly it was felt generally that acquired aortic stenosis, especially when associated with calcification, was usually of arteriosclerotic origin,[8] today, most authors feel that the majority are caused by rheumatic valvulitis.[9,10] In our experience, approximately one half of the patients with acquired aortic stenosis also present coexisting indisputable rheumatic disease of the mitral valve. Presumably, both valves in this group of patients have been affected by the same etiological process. Furthermore, since the majority of the remaining patients present a spectrum of aortic pathology which is identical with that observed in combined aortic and mitral valvular disease, we are inclined usually to indict the rheumatic state for the initiation of acquired aortic stenosis. The frequency of the occurrence of calcification in the involved valve is not pertinent since calcification develops occasionally in congenitally deformed, and, very often, in rheumatic stenotic valve lesions. It is found to a greater or lesser extent in over 90 per cent of stenotic aortic valves examined digitally during transaortic surgery or at autopsy.

In general, we have felt that the arteriosclerotic process does not produce commissural fusion but merely stiffens the nondeformed valves in a position of incomplete closure[13] (Fig. 2). Thus, some insufficiency must coexist in practically every one of these valves.

Aortic Stenosis Resulting From Subacute Bacterial Infection.—While such lesions undeniably exist due to the organization of the vegetations and the fibrosis which ultimately may develop upon the healing ulcerative lesions, they are relatively rare. Since bacterial infection apparently always must be superimposed upon a valve already deformed by congenital malformation or rheumatic distortion, the structural abnormality tends to follow the pattern of the primary pathology and is amenable to therapy appropriate for it.

Rheumatic Aortic Stenosis.—The pathogenesis and pathology of rheumatic aortic stenosis are in most respects analogous to that of rheumatic mitral stenosis, so well described by Rokitansky[14] and Magarey.[15] It appears to occur clinically about one-fifth as frequently as the latter disease, and shows a preponderant predilection for males. Aortic stenosis was diagnosed in 365 (32.5 per cent) of 1,585 patients operated upon for rheumatic valvular disease at the Hahnemann Hospital in Philadelphia. Rheumatic vegetations developing upon the free margins and the lunulae (zones of leaflet contact) of the cusps produce surface irregularities upon the ventricular aspect of the valve. Blood flowing through the valve passageway develops a peripheral turbulence due to this roughening of the walls of the channel. In spite of the rapid blood flow, a certain amount of surface deposition of platelets and fibrin results (perhaps related in part to defibrination of the flowing blood). Organization, both of the vegetations (really minute thrombi) and of the fibrinous deposit upon the valve, applies a thin layer of scar tissue upon the ventricular aspects of the cusps and over their free margins. Until the surface irregularities have become buried completely within additional subsequently laid down lamellae of scar tissue and incorporated within the thereby thickened valve substance, the process must recur from time to time even in the absence of any further rheumatic exacerbation. Obviously, an exacerbation of the rheumatic state may reproduce a new cycle of surface irregularity, turbulent flow, fibrinous deposition, organization, etc. Thus, "new" tissue of fibrous character becomes added to the ventricular aspect of the leaflets. To a lesser extent, the process may "spill over" onto the concave aspect of the valve and bring about thickening of the aortic surfaces of the cusps.

At the same time, the fibrinous exudate, due to the incessant movement of the cusps, tends to become heaped up and to bridge the most peripheral extremities or angles formed by the adjacent cusps at their aortic attachments. As this fibrin becomes organized, the peripheral portions of the free margins of the cusps become adherent. Ultimately, a centripetally progressive adhesive valvulitis results in extensive cross fusion of the free edges and the lunulae of the leaflets (Fig. 3, *A*, *B*, *C*, *D*, *E*).

Logically, this process, if symmetrical, gradually should reduce the valve orifice to a tiny central equilateral triangular opening However, progressive *centripetal cross fusion of the leaflets may advance more rapidly in one or two* commissures while the remaining two or one may be less involved Thus, a bicuspid transformation of the valve may take place, or but a single slitlike radiating aperture may persist (Fig 4, *A*, *B*, *C*, *D*) Commissural obliteration (cross fusion of the adjacent free margins of the cusps) beginning at the base of the valve is the most characteristic gross anatomic feature of rheumatic stenosis occurring in the aortic valve (as well as of the mitral and tricuspid)

Late contraction of the accumulated lamellae of scar tissue which have been laid down upon the free margin of the reduced valve orifice may lead to a secondary "purse string" type of narrowing of the central (or eccentric) residual opening Thus, the initial stenosis, due to the commissural obliteration, may become even tighter On the other hand, the major line or direction of placement of the strands of scar tissue applied to the surface of the valve may be such that the preponderant late effect is retraction of the thickened margins of the cusps toward their basal attachment Again, this may be either *a symmetrical or an asymmetrical phenomenon* Thus, accurate diastolic coaptation of the apposed valve edges may become impossible, causing a greater or lesser degree of incompetence to become superimposed upon the primarily stenotic lesion Not infrequently, the clinical evidences of such regurgitation may come to overshadow those of a dynamically less signifi stenosis (Fig 5, *A*, *B*)

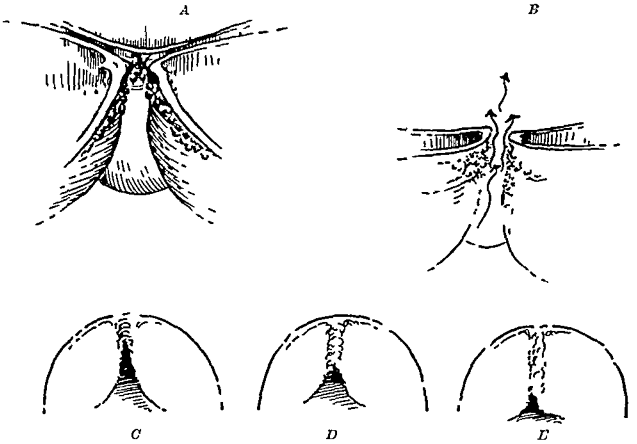
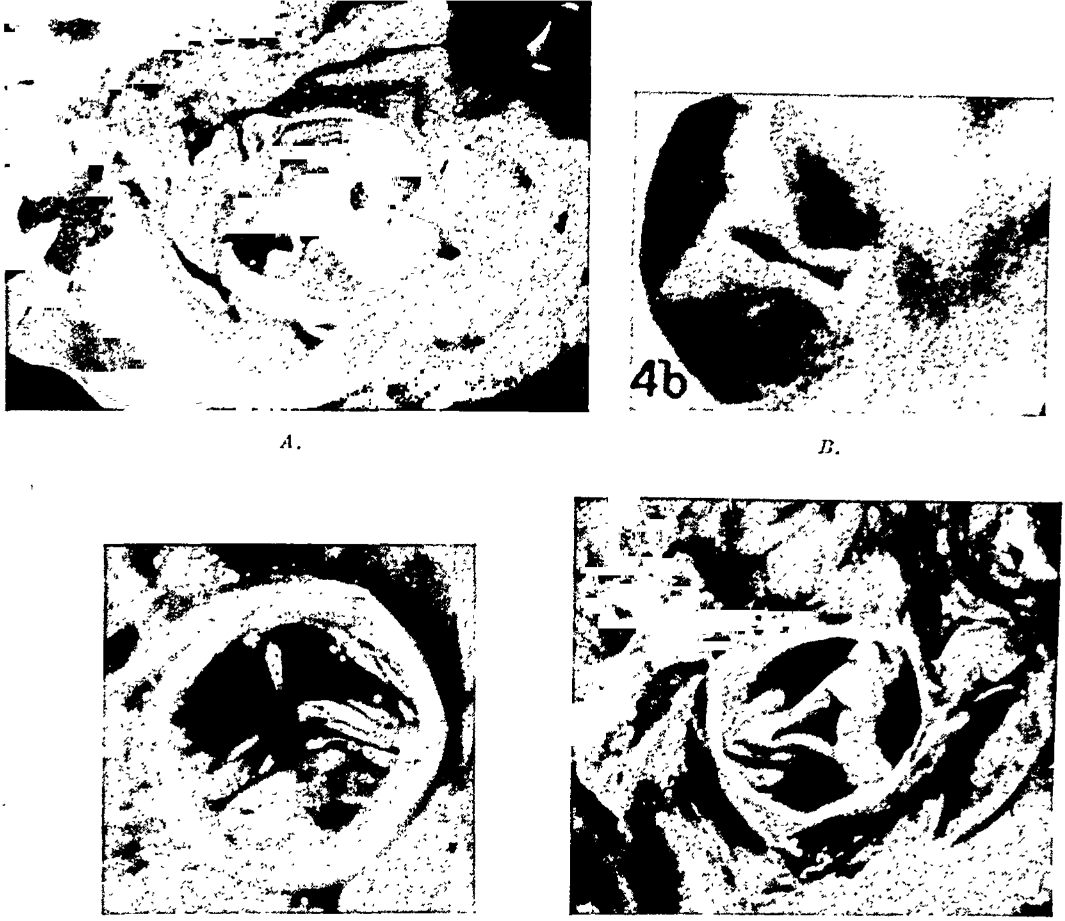


Fig 3—*A* Early appearance of vegetations upon the zones of leaflet approximation in rheumatic aortic valvulitis This localization may be related to the repeated trauma of valve closure *B* The surface irregularities produced by these vegetations engender a turbulence of flow which leads to recurrent deposition of fibrin and platelets upon the ventricular aspect of the valve *C* Beginning cross fusion of peripheral portions of adjacent leaflets due to organization of heaped up fibrin *D*, Centripetally progressive obliteration of valve commissure *E* Final state of nearly complete commissural obliteration (From Bailey Surgery of the Heart courtesy Lea & Febiger)



A. B.

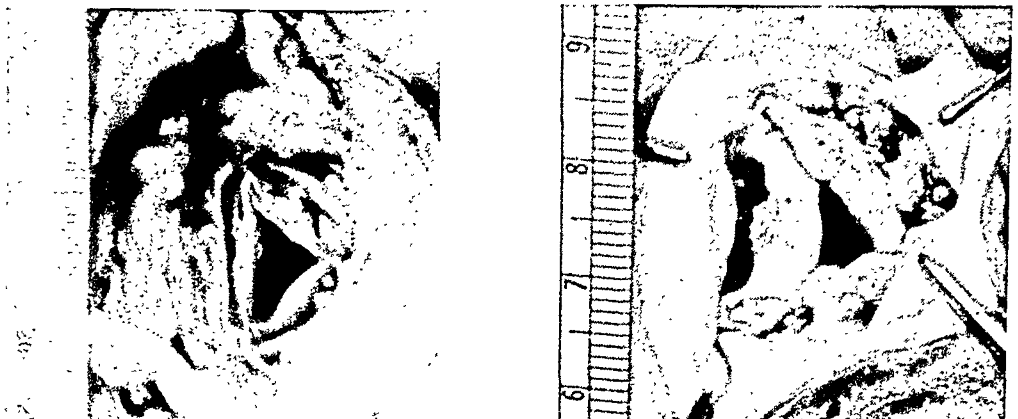
C. D.

Fig. 4.—*A*, Photograph showing early stage of rheumatic aortic stenosis. Note beginning symmetrical obliteration of the commissures. (Courtesy Dr. Raymond Truex, Hahnemann Hospital, Philadelphia.)

B, Advanced aortic stenosis showing complete obliteration of two commissures, a radiating slitlike opening representing the one remaining unfused commissure. (From McMillan, Aortic Stenosis: A Post-mortem Cinephotographic Study of Valve Action, Brit. Heart J. 17: 56, 1955.)

C, Complete obliteration of one commissure while the others remain open. This results in bicuspid transformation of the valve. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

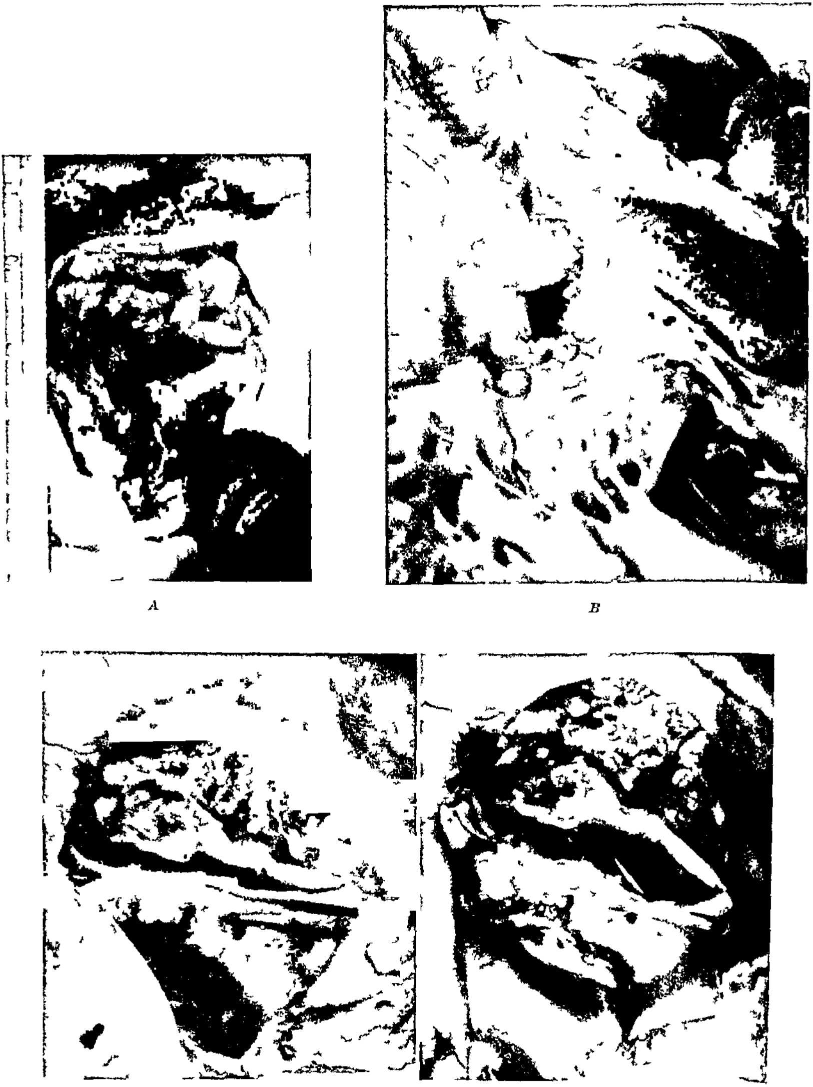
D, Extensive symmetrical obliteration of all three commissures reduces the valve opening to a diminutive central triangular passage. (From Gould, Pathology of the Heart, courtesy Charles C Thomas.)



A. B.

Fig. 5.—*A*, Retraction of central edges of cusps in rheumatic aortic valvulitis brings about an element of aortic insufficiency which sometimes may overshadow the coexisting stenosis. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

B, Aortic regurgitation is often found in association with stenosis when the valve leaflets are extensively calcified. In this case, the insufficiency was not dynamically significant. (From Larzelere and Bailey, Aortic Commissurotomy, J. Thoracic Surg. 26: 66, 1953.)



A

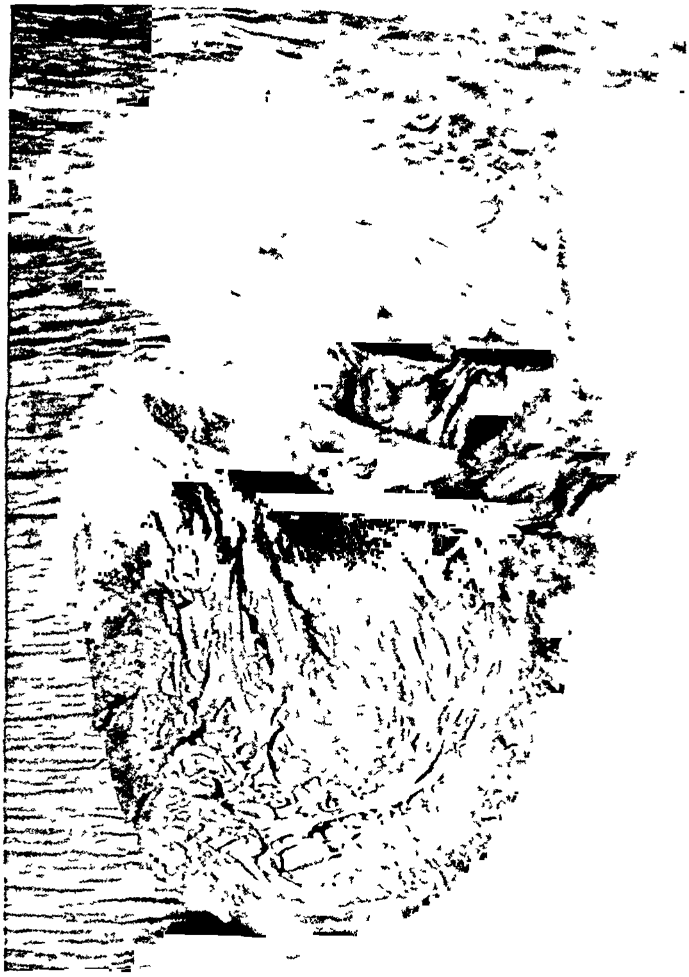
B

C

D

Fig 6—4 Extensive calcification of the aortic valve seen from the arterial aspect Note irregular excrescences extending up the wall of the aorta
B Ventricular aspect of an extensively calcified aortic valve This calcification may extend into the septal cusp of the mitral valve as well as into the interventricular septum
C, Clamshell like aortic valve presenting extreme calcification and only a very tiny slitlike residual orifice (toward the right of the photograph) (From Bailey Surgery of the Heart courtesy Lea & Febiger)
D Forcible instrumental separation of valve seen in Fig 6 C Note limitation of possible restitution of valve movement due to extreme rigidity of it 4 (From Bailey Surgery of the Heart, courtesy Lea & Febiger)

With the prolonged immobilization of the thickened leaflets, both due to intrinsic stiffening and to fixation to *adjacent cusps, late degenerative changes* are prone to develop. This may account, in part, for the very great incidence of calcification of the leaflets observed in aortic stenosis. However, it is probable that the relatively high pressures developed within the left ventricle and aorta and their violent fluctuations with consequent resulting trauma to the valve also are responsible, at least in part, for the greater incidence of calcific changes observed in this valve than in others involved by a similar stenotic process. Thus we may explain the relative rarity of calcific changes in stenotic right-sided heart valves (tricuspid and pulmonic) which are subjected essentially to much lower pressures. In mitral stenosis where there is impaired ventricular filling with consequently less trauma to the valve structures, the incidence of calcific changes is of intermediate frequency (38 per cent). Characteristically, calcification of the aortic valve seems to involve the region of the obliterated commissures even more frequently and more extensively than the



A.

Fig 7, *A.*—*Photograph of autopsy specimen showing extreme thickening of left ventricular wall in aortic stenosis In pure forms of this lesion, there is no enlargement of the ventricular chamber. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger)* margins and fundi of the cusps themselves. Not infrequently, the calcification may extend from the commissures into the adjacent aortic wall, and may encircle it to form a complete calcific ring. Occasionally, the calcification extends up the

B.

(or brachial arterial) pressure curves. Apparently, they must be performed with the patient unanesthetized and with an intact chest wall since such determinations obtained directly during surgery usually present a significant "physiologic damping" (Fig. 11, *A*, *B*). Probably this phenomenon is related to a reduction in stroke (and minute) output. For the same reason it is important to measure the cardiac output by simultaneously performing right and left heart catheterization.

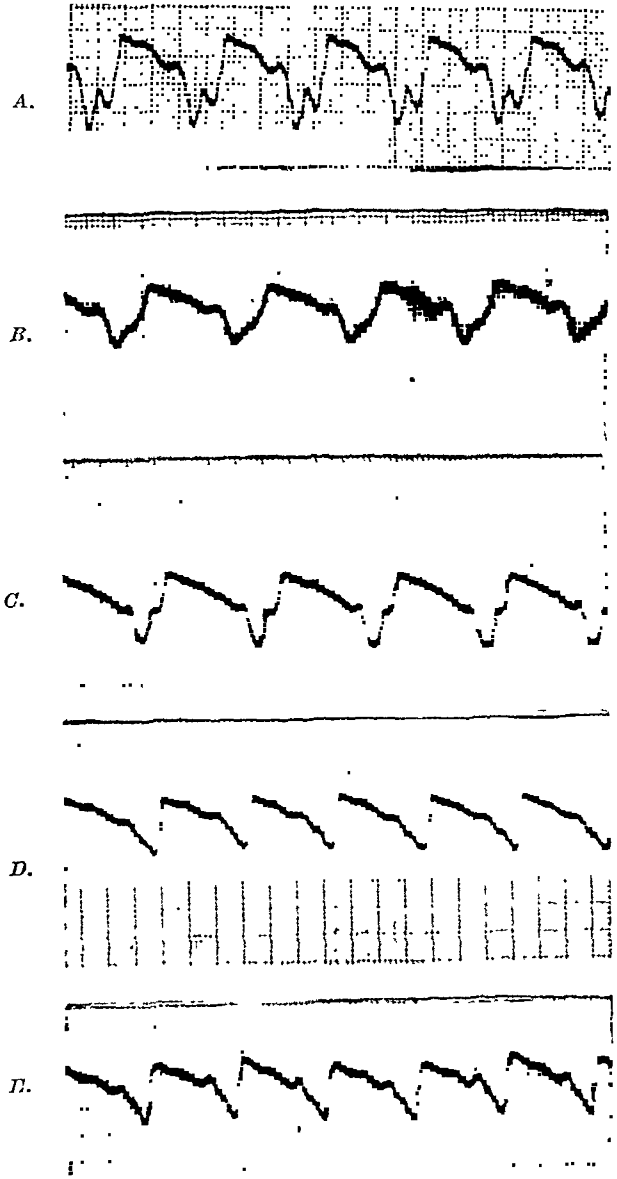


Fig. 10.—*A*, Normal brachial arterial pressure tracing. *B*, *C*, *D*, and *E*, Four patterns of the brachial arterial pressure curves which suggest the existence of a significant degree of aortic stenosis. (From Goldberg, Bakst, and Bailey, The Dynamics of Aortic Valvular Disease, Am. Heart J. 47: 527, 1954.)

Another feature characteristic of severe aortic stenosis is the development of coronary insufficiency. While, undoubtedly, intrinsic coronary arterial disease sometimes may coexist, in most cases the insufficiency is due entirely to the altered kinetics of flow. Support for this opinion is obtained from the usual finding of anatomically normal coronary arteries at autopsy in individuals who have succumbed from stenotic aortic disease and who have manifested a severe anginal syndrome. Also, it is strengthened by the nearly constantly observed relief of angina, when preoperatively marked, following successful aortic commissurotomy.

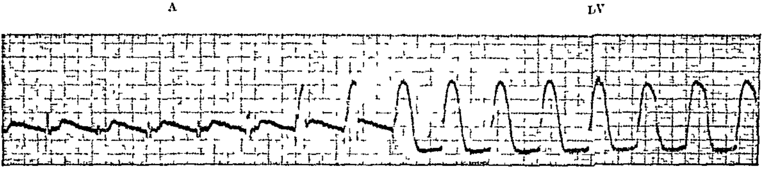


Fig 11, A

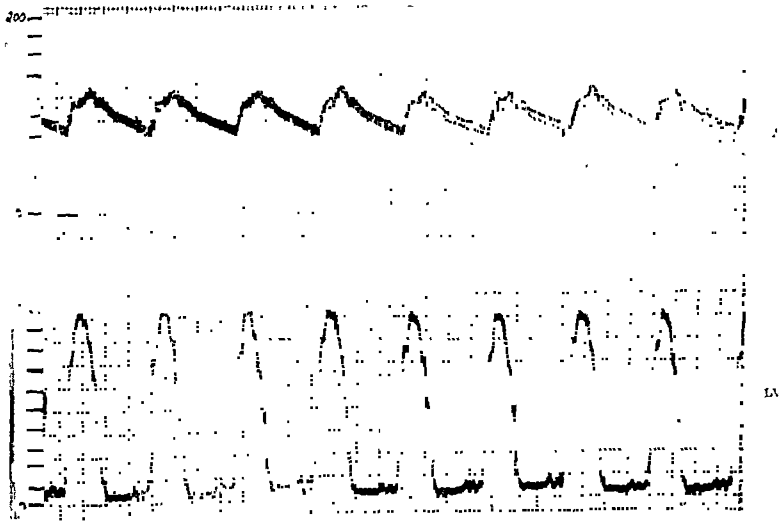
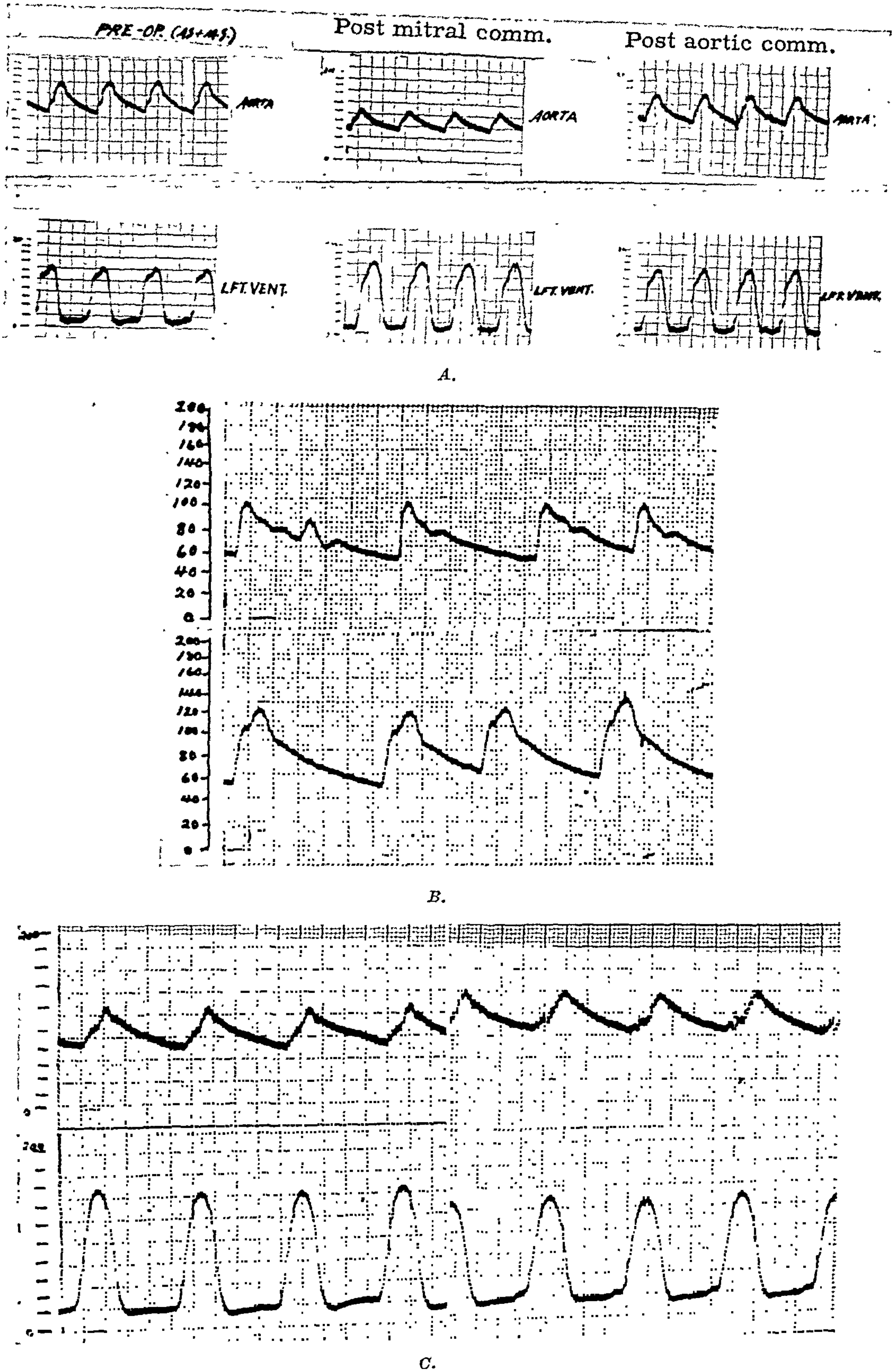


Fig. 11, B.

--tic (A) and left ventricular (LV) pressure tracing obtained
by ca· › of the heart (transthoracic approach). The range is 400
mm. radient of approximately 120 mm. Hg. B, Simultaneous
direct om the aorta (A) and left ventricle (LV) in the same patient
after definitive maneuvers. Since the range is 200 mm Hg, the
gradie Hg. Apparently significant alterations in physiology de-
velop ry which render impossible accurate correlation with studies
performed under other conditions

It seems probable that the over-all lowering of the pressure at the root of the aorta is associated with a corresponding reduction in coronary flow. Green[22] feels that this reduction occurs largely during systole ar
by the relatively greatly increased peripheral c arterial



A.

B.

C.

Fig. 12, *A. B,* and *C.* *(For legend see opposite page.)*

"boot-like" configuration of the radiographic shadow will be seen. Calcification of the aortic valve is best seen on fluoroscopic examination and was demonstrated in one-half of our cases.

MULTIVALVULAR DISEASE

In patients exhibiting aortic stenosis, either "pure" or combined with a dynamically insignificant degree of aortic insufficiency, one not infrequently finds coexisting mitral valvular disease. Forty-four per cent of our patients of this group were found to have significant mitral stenosis, with or without varying degrees of mitral insufficiency. Five and nine-tenths per cent manifested evidence of mitral insufficiency alone. The majority of this latter group presumably were suffering from dilatation of the mitral annulus fibrosus with resulting functional insufficiency secondary to the left ventricular enlargement caused by the aortic lesion.

In these individuals with combined aortic and mitral valvular disease, one must always consider whether the tricuspid valve may not also be affected Tricuspid valvular disease was found in 17 per cent of the patients with mitral and aortic rheumatic valvular disease. Predominant tricuspid stenosis was recognized by direct digital examination in 11 per cent of patients with both mitral and aortic valvular disease. It appears that acquired tricuspid stenosis practically never is found as an isolated lesion. It seems always to be accompanied at least by a similar mitral valvular lesion.

Apparently, rheumatic or other acquired stenosis of the pulmonary valve is extremely rare. Acquired pulmonary insufficiency (usually functional) apparently is a lesion of relatively minor clinical significance.

The observed occurrence of significant disease of one or both atrioventricular valves in association with aortic valvular disease is of great importance since failure to relieve all physiologically important lesions may spell failure to obtain clinical improvement or even immediate disaster to the patient. It is a well-accepted hydrodynamic principle that one unrelieved point of serious obstruction in a fluid flow system will impede the passage of the current almost as much as two or more similar points of obstruction, since the limited volume passing through one such barrier can easily traverse the next one.

On the other hand, relief of an early valvular obstruction without simultaneous relief of one farther on in the circulation will have the effect of aggravating the manifestations of this later one. Thus, if mitral stenosis is relieved without concomitant commissurotomy for any significant associated aortic

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Fig. [illegible] taneous aortic (upper) and
pressure [illegible] surgery in a patient with cor
stenosis [illegible] obstruction, the gradient betw
increased [illegible] pressure curve develops the
stenosis [illegible] anacrotic slurring are abolishe
suing aor [illegible] Bailey, Surgery of the Heart.
B, Upper, Brachial arterial pressure curve obtained preoperatively
stenosis who was considered clinically to have only very mild aortic stenosis Lower, Brachial tracing taken after mitral commissurotomy showing the typical anacrotic notch of aortic stenosis Physiologically the aortic stenosis had become dynamically significant because an increased amount of blood was now being expelled through an aortic aperture of unchanged size *C*, Abolition of systolic pressure gradient obtained between the left ventricle and the aorta in a patient with isolated aortic stenosis Left, Precommissurotomy tracings Right, Tracings obtained immediately after aortic commissurotomy

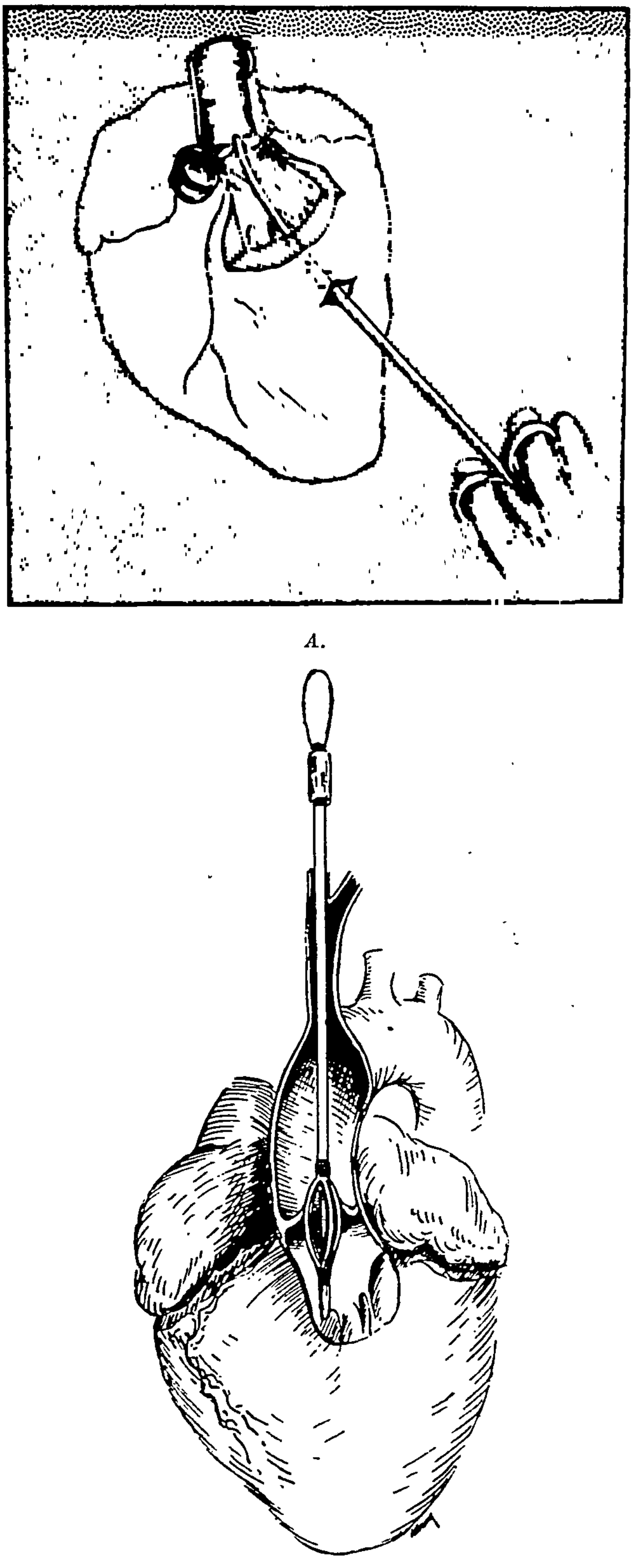

Fig. 13, *A* and *B*.—*A*, Earliest attempt at surgical division of stenotic aortic value by a odification of Brock's method of pulmonary valvulotomy. The patient did not survive. *B*, ubsequent attempt at dilatation of the stenotic aortic valve by retrograde passage of an strument with heavy umbrella-spring type blades. The patient survived. (From Larzelere nd Bailey, J. Thoracic Surg. 26: 66, 1953.)

stenosis, the clinical importance of the latter becomes enhanced. This is due to the improvement in left ventricular filling which requires this chamber to eject a greater quantity of blood through the same stenotic aortic orifice. Hence, relatively speaking, the aortic stenosis becomes aggravated physiologically. Since isolated aortic stenosis characteristically follows a more malignant course than does the combination of mitral and aortic stenosis, it is evident that such "conservative" treatment must be a disservice to the patient (Fig. 12, *A*, *B*, and *C*).

SURGICAL CONSIDERATIONS

Tuffier[24] in July, 1913, attempted digitally to dilate a stenotic aortic valve in a young male adult by invaginating the dilated anterior aortic wall at a point just above the valve level. The patient survived at least 10 years and was reported improved. Smithy[25] attempted to resect a portion of a stenotic aortic valve in the summer of 1948. He intended to use a modified sphenoid punch inserted through a transventricular approach. Unfortunately, the patient expired suddenly during the induction of anesthesia, apparently due to the onset of acute ventricular fibrillation.

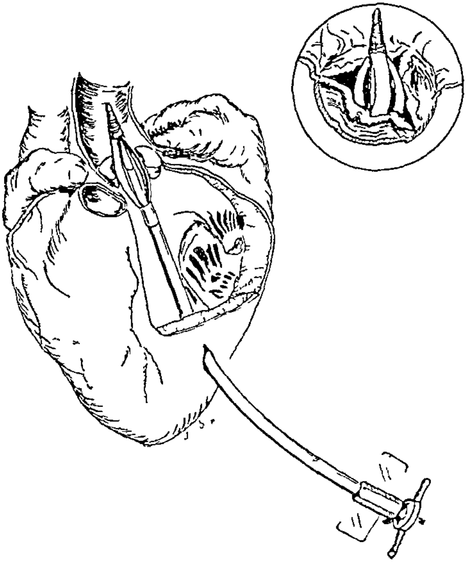

Fig. 13. *C*.—Curved instrument with similar dilator mechanism subsequently used by transventricular approach.

On March 9, 1950, the senior author (C. P. B.)[26] attempted to divide a stenotic aortic valve in a 26-year-old white housewife using a modification of Brock's[7] method of pulmonary valvulotomy and a transventricular approach. Although the valve was divided according to plan, death on the operating table resulted, due to the creation of a significant degree of insufficiency. This unfortunate experience confirmed certain theoretically-based fears that the sudden production of aortic regurgitation would not be tolerated well in a patient who had become adjusted or "compensated" to a severe degree of aortic stenosis. It was felt, thereafter, that neither cutting across nor excision of a portion of an aortic valve cusp could be justified clinically.

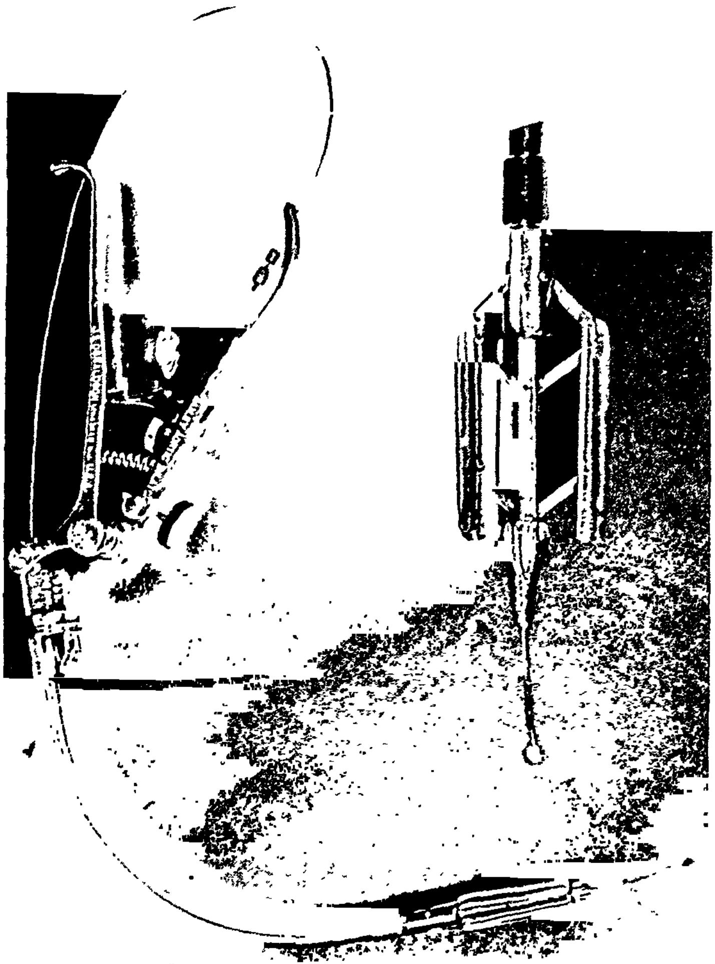

Fig. 14.—Modern triradiate parallel bar type of aortic dilator. Note the incorporated guide wire and olivary tip. The entire head is designed for free rotation on its shaft. This assures accurate automatic alignment of the triangular cross-sectional contour of the open or closed dilator with the outline of the diminutive valve orifice if any remnant of its initial triangularity persists. (*From Bailey, Surgery of the Heart, courtesy Lea & Febiger.*)

Experimental efforts to "dilate" the stenotic aortic valve in the autopsy room revealed that the fused commissures tended to separate on application of disruptive pressure more readily than did the continuity of the sclerosed leaflets. Hence, it became evident that surgical aortic commissurotomy was a logical possibility. At first, it was felt that this must always be an instrumental accomplishment since it was not then foreseen that deliberate digital examination and manipulation of the valve during life would become feasible.

On April 6, 1950, one of us (C. P. B) performed an instrumental dilatation of a stenotic aortic valve by passing an "umbrella-type" dilator through the valve orifice in retrograde fashion, guiding it through an incision made in the right common carotid artery and through the ascending aorta to the valve orifice which was located by instrumental palpation. While this particular operation was considered successful (with survival), subsequently the procedure was abandoned in favor of a somewhat similar one performed by a transventricular route (Fig. 13, *A, B, C*).

By April, 1952, in large part due to the combined efforts of our engineering associate, Major J. Shearman Donaldson,* and our chief surgical resident, Dr. Henry B. Larzelere,** a much superior instrument was developed for this purpose (Fig. 14). In it, the umbrella-spring type blades have been replaced by three parallel dilating bars with wedgelike cross sections. A retractable guide wire with an olivary tip has been incorporated within the instrument to serve a function comparable to that of the filiform bougie used in the practice of urology. An incorporated rotatory mechanism permits free turning of the dilating head upon its shaft for automatic adjustment of its triangular cross section (open or closed) to the usual residual triangularity of the stenotic valve orifice. Thus, the unsharp edges of the dilating bars tend to become aligned accurately against the respective angles of the valve orifice. Since these angles represent the central extremities of the fused commissures, mechanical separation of the bars applies parting or splitting pressure directly and most effectively against the commissures, thus tending to separate and mobilize the three functional valve elements anatomically. It was realized that in many valves extreme and perhaps asymmetric sclerosis or calcification might prevent complete separation of all commissures, and that indeed it might be accomplished only partially, perhaps in only one or two of the three. However, it was reasoned that any measure of anatomic separation accomplished would represent at least a partial restoration of a more normal valvular state and would result in a degree of functional improvement. To obtain the desired effect, it was deemed essential that at least some valvular or aortic wall flexibility remain. Since the left ventricle would, in these very cases, be maximally adjusted to compensate for any residual unrelieved aortic stenosis, enlargement of the orifice to more than one-quarter of its normal size would result

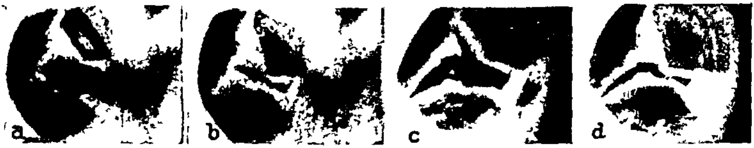

FIG 15.—*a*, Stenotic aortic valve orifice revealing only one unobliterated commissure—systolic position *b*, Same valve in diastole (closed) *c* After passage of triradiate aortic dilator, all three commissures have become separated, and some normal type valve action is provided Systole *d*, Closure of valve indicates that competence is fully maintained (From McMillan, Brit Heart J. 17: 56, 1955.)

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*Major J Shearman Donaldson, Chatham, N J.

**Dr. Henry B Larzelere, Toledo, Ohio

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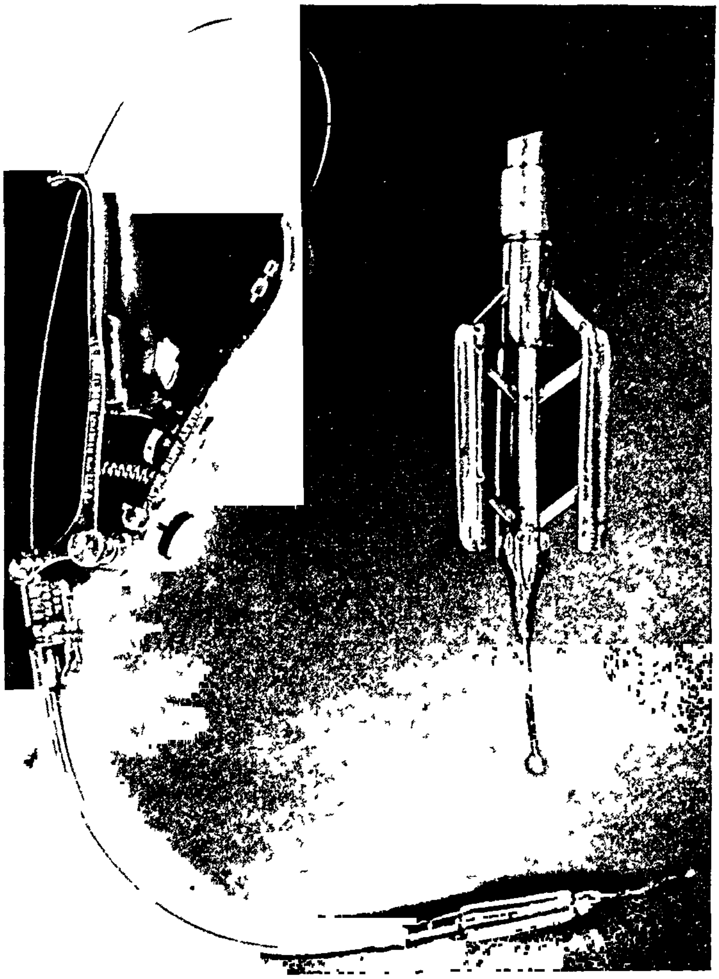

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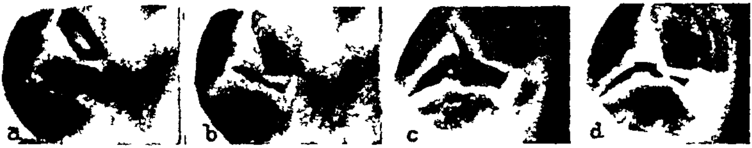

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*Major J Shearman Donaldson Chatham N J
**Dr Henry B Larzelere Toledo Ohio

essentially in full clinical relief. This concept depended for its validity upon the maintenance of a state of valvular competence at least the equal of that existing preoperatively (Fig. 15, *A, B, C, D*).

In large measure, these concepts and hopes have been justified by our subsequent clinical experiences. On April 4, 1952, the improved instrument was used with great initial success (by the transventricular route) in a male patient who was nearly moribund. He attained an excellent state of improved health and maintained it for two years subsequently. Ultimate recurrence of symptoms with an unusually rapid downhill course precluded a second surgical attempt. Since then, 211 patients with prominent evidence of aortic stenosis have been operated upon by this instrumental technique, most of them obtaining excellent clinical and physiologic improvement. When mitral stenosis has coexisted, as it did in over 40 per cent, mitral commissurotomy was carried out simultaneously by way of the usual left appendageal approach. However, the operative mortality (Table I) associated with this technique has remained relatively high.[13, 28, 29] Daniel and Scott,[30] Muller and Hyman,[31] and McMillan[17] have reported similar experiences. In order to eliminate nonrelevant considerations such as those related to the presence of a major element of aortic or mitral insufficiency, or of coexisting tricuspid disease, we have tabulated the operative results only in those patients considered to have preponderant aortic stenosis and those with combined preponderant aortic and mitral stenosis (Tables I and II).

TABLE I. TRANSVENTRICULAR APPROACH FOR AORTIC COMMISSUROTOMY SHOWING EARLY OPERATIVE RESULTS

TYPE OF LESION	NUMBER OF CASES	NUMBER OF OPERATIVE DEATHS	OPERATIVE MORTALITY (%)
Aortic stenosis	37	9	24.2
Aortic stenosis with insignificant aortic insufficiency	31	10	32
Total	68	19	28

TABLE II. OPERATIVE RESULTS IN COMBINED SIMULTANEOUS AORTIC AND MITRAL COMMISSUROTOMY IN PATIENTS WITH AORTIC AND MITRAL VALVULAR DISEASE (TRANSVENTRICULAR AND LEFT AURICULAR APPROACHES)

TYPE OF LESION	NUMBER OF CASES	NUMBER OF OPERATIVE DEATHS	MORTALITY (%)
Aortic stenosis plus mitral stenosis	22	1	4.5
Aortic stenosis plus mitral stenosis with mitral insufficiency	10	2	20.0
Aortic stenosis with aortic insufficiency, plus mitral stenosis	24	4	16.7
Aortic stenosis with aortic insufficiency, mitral stenosis with mitral insufficiency	31	9	29.0
Total	87	16	18.4

Consideration of the various ways by which this risk might be reduced has led to some interesting speculations. Breaking down the over-all series of cases into pertinent smaller groups (Tables I and II) revealed that patients with combined aortic and mitral stenotic lesions simultaneously corrected apparently ran less risk than those with essentially "pure" isolated aortic stenosis in whom the aortic valve alone was operated upon. Apparently, there is a dynamic difference in the way isolated aortic stenosis and combined aortic and mitral stenosis affect the heart and circulation.

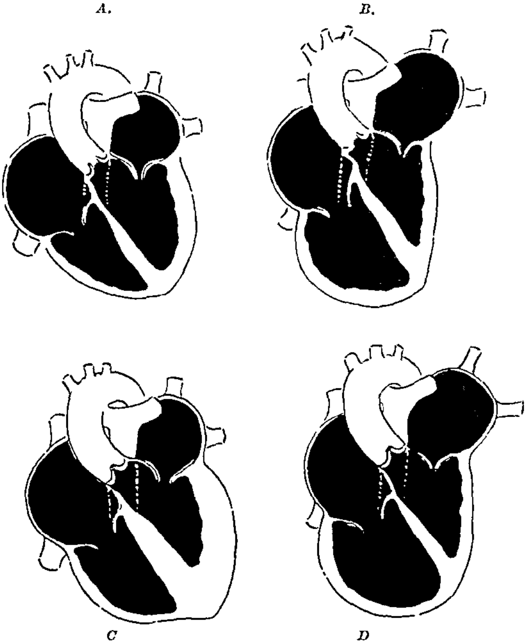

Fig 16—Diagrammatic representation of respective types of chamber involvement with various types of valvular stenosis *A*, Normal heart *B*, Heart in isolated mitral stenosis Note the dilated right ventricle and left atrium and the small left ventricle—this latter chamber is protected by the inlet stenosis against overwork or strain *C*, Heart in isolated aortic stenosis Note the tremendous concentric type of hypertrophy of the left ventricle without dilatation of the chamber resulting from the overwork consequent to the severe outlet stenosis in the presence of normal filling (no inlet stenosis) The left atrium and right ventricle remain normal until left ventricular failure is imminent *D*, Heart in combined mitral and aortic stenosis Note similarity to condition found in isolated mitral stenosis The right ventricle and left atrium become dilated and overworked but the left ventricle remains small and relatively healthy It is protected against the effects of outlet obstruction by the severe inlet, stenosis The small amount of blood entering the left ventricle is readily expelled through the reduced aortic outlet.

Fig. 16 indicates the different types of chamber involvement seen in these various states of aortic or mitral stenotic valvular disease. It is evident that in isolated aortic stenosis the left ventricle bears the full brunt of the disease, becoming overworked and hypertrophied due to its outlet obstruction. As has been mentioned previously, it has been observed that the left ventricular wall in this condition tends to become soft and degenerated, and hence to hold sutures with difficulty. This tendency for sutures to cut through the soft myocardium was responsible for at least two deaths from hemorrhage, as indicated in Table III.

TABLE III. CAUSES OF DEATH IN PATIENTS OPERATED UPON FOR AORTIC STENOSIS BY TRANSVENTRICULAR TECHNIQUE

CAUSE OF DEATH	NUMBER OF DEATHS
Failure, unclassified	1
Cardiac arrest	5
Ventricular fibrillation	5
Hemorrhage, uncontrolled	2
Cerebral embolism	3
Hypotension (after leaving operating room)	1
Hypotension (in operating room)	1
Cardiac arrest Ventricular fibrillation Laceration of coronary artery	1
Total	19

Furthermore, the hypertrophied left ventricle in these patients very often exhibits hyperirritability tending on relatively slight stimulation to develop runs of ventricular extrasystoles or even ventricular fibrillation. A number of deaths which occurred in the operating room were due to the latter arrhythmia (Table III). Cardiac asystole, perhaps related to the myocardial degeneration, also was a prominent cause of death in this series.

In contrast, the left ventricle in patients with combined aortic and mitral stenosis (see Fig. 16, *D*) characteristically is small and of firm structure readily capable of holding hemostatic sutures securely. It exhibits little or no evidence of unusual irritability to mechanical stimulus. In general, at operation the heart in combined aortic and mitral stenosis assumes the configuration of that seen in pure mitral stenosis, presenting a large right ventricle, pulmonary artery, and left atrium, with a normal-sized or small left ventricle. Of course, it is understood that varying intermediate proportions of these effects may be observed with various degrees and combinations of these lesions.

Post-mortem examination in several of the operated patients showed that a degree of aortic insufficiency inadvertently had been produced during the surgical procedure. In at least two instances, a valve orifice which had developed a bicuspid configuration had resisted tricommissural separation with resulting laceration of the anatomic continuity of a sclerotic cusp. In another, an indescribably distorted calcareous valve had become irregularly fractured, probably resulting in insufficiency. Likoff and associates[29] have reported a 13 per cent incidence of clinically increased aortic insufficiency in patients surviving instrumental transventricular aortic commissurotomy. Varying degrees of regurgitation were created in 5 per cent of the cases without detectable insufficiency prior to surgery.

Because of these anatomic variations, it seems probable that there will always remain a certain irreducible incidence of created aortic regurgitation in purely instrumental attempts at tricommissural separation of the stenotic aortic valve. How much influence such inadvertent creation of insufficiency had upon those deaths listed under the terms "cardiac arrest," "hypotension," "renal failure," "heart failure," and "ventricular fibrillation" cannot be estimated with accuracy. However, deaths from such causes have become significantly rare since the development and adoption of the now preferred transaortic digitally guided technique of performing aortic commissurotomy.

After due consideration and reflection, it was concluded that, at least in cases of isolated aortic stenosis, the development of some approach to the *stenotic aortic valve other than the transventricular one was mandatory.* It was deemed of paramount importance that direct digital examination and guidance of instrumentation be incorporated into the procedure if at all possible. McMillan[17] recently has voiced a similar hope. Since both Brock[32] and the senior author (C. P. B.)[26] already had had previous bitter experiences in attempting to approach the aortic valve by way of the left atrium and the mitral orifice, our attention was directed toward the ascending aorta as a possible avenue of approach.

Experiences with direct incision of the aortic wall in the experimental laboratory regardless of the use of various hemostatic devices convinced us that a better means of entrance into its lumen must be devised. Vascular homograft segments sutured to the aortic wall proved unsatisfactory largely because of the fragility of their structure. However, the use of pericardium to form a pouch or "appendage" upon the aorta proved reasonably satisfactory, and gradually a suitable technique for its application was developed. For the first time, it became possible methodically to palpate the aortic valve and to manipulate it under the guidance of "digital vision" without any greater difficulty or disturbance to the cardiac action than is experienced in comparable exploration and manipulation of the mitral valve.

On April 4, 1953, the first successful digitally guided aortic commissurotomy by the supravalvular route was performed by one of us (C. P. B.), using a pouch prepared from the patient's own pericardium. On that occasion, valvular dilatation was accomplished by inserting the three-bladed aortic dilator through a second opening made in the pouch and guiding it through the valve with the intravascular finger.[33] Since that time 61 patients have been *operated upon by this method. In 40 per cent of the cases, a satisfactory commissurotomy* has been accomplished by digital pressure only. In the remainder, instrumental assistance has been required.

A two-bladed dilating instrument has been designed* to be worn on the finger and inserted with it through the pouch and into the aorta (Fig. 17). Its two nonsharp wedgelike blades separate at a 110-degree angle to part simultaneously those two commissures which, at that time, the intra-aortic finger is able to feel. By the sense of touch, the finger *readily can follow the splitting* effect upon the valve, and so is able to limit or control it.

We have tried latex, fabric, and plastic pouches, as first suggested by Glenn[34] and later by Swann,[35] but still prefer pericardium for this purpose. Pericardial homografts may be prepared and preserved by the method of

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*Obtainable through George P. Pilling & Son Company, Philadelphia, Pa

Meeker and Gross.[36] Their use obviates the necessity for removing most of the patient's own natural pericardial support, and also for transecting the sternum and opening both pleural cavities.

As an alternative method, plastic fabric may be used to fashion an aortic pouch. Of the various materials of this type we have tried, Vinyon fabric* has proved most satisfactory, mainly because of its elasticity and lack of tendency to fraying. However, a square of nylon cloth may be used provided the cut edges (peripheral as well as about the central incision) are flashed momentarily through the flame of a lighted match. The heat melts the plastic slightly and seals the cut edges. With both fabrics we have found a technique similar to that employed with the pericardial homografts reasonably satisfactory.

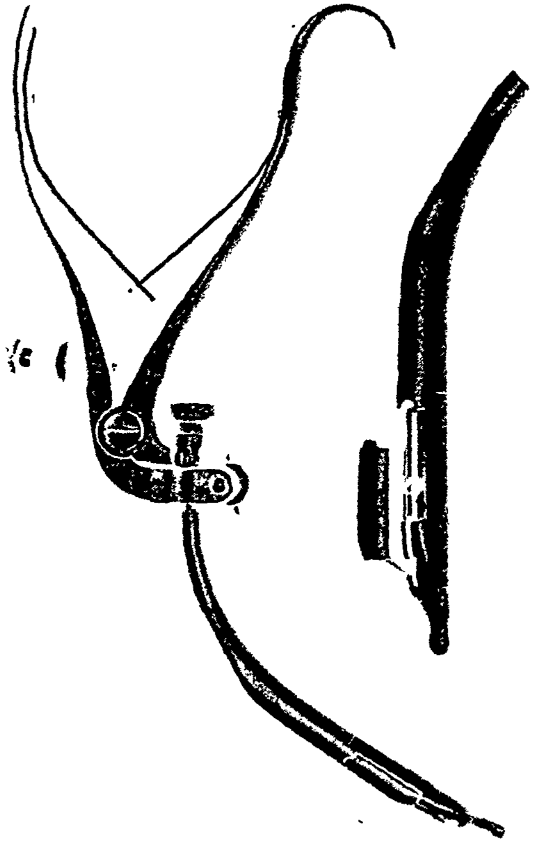

Fig. 17.—Two-bladed aortic dilator designed to be worn on the operator's finger and inserted with it through an artificially created aortic pouch sutured to the artery to permit an approach to the valve in retrograde fashion. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

At the suggestion of one of the authors (H. E. B.), this operation has been modified so that it can be carried out satisfactorily through a right anterior intercostal incision only (third interspace). With one intact pleural space (left) and an undivided sternum, an effective cough mechanism is maintained, and thus the postoperative problems of evacuating the bronchial secretions are minimized.

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*Obtainable from American Viscose Corp., Marcus Hook, Pa.

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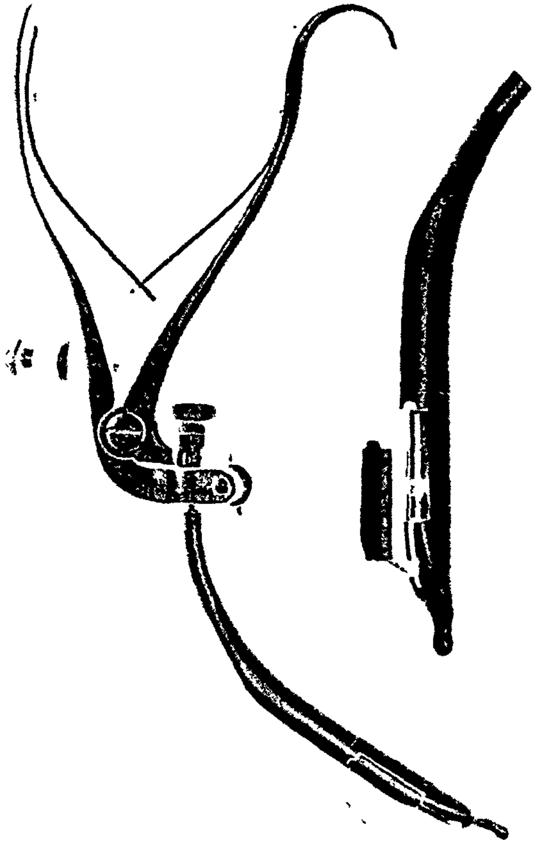

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At first, it was not deemed feasible to operate simultaneously for a coexistent mitral stenosis either from the right side or by way of a left anterior intercostal approach, with the patient placed in the supine position preferred for transaortic commissurotomy. Therefore, in view of the observed lower operative risk with coexistent *aortic and mitral stenosis* (Table II), it was advised formerly that the transventricular route to the aortic valve be chosen in patients with combined mitral and aortic stenosis.[13, 28, 33] Since the lateral posture usually was employed for the aortic dilatation, the mitral valve was readily approached by way of the left auricular appendage either just before or just following the definitive aortic surgery.

However, certain unsatisfactory operative results obtained in such combined cases, and the development of a satisfactory approach to the left atrium and mitral valve by way of the dissected interatrial groove[13, 37] have caused the authors to alter this expressed opinion.

Indeed, the experience and satisfaction obtained in operating from the right side for mitral stenosis combined with aortic stenosis have since led us to choose this approach electively in many patients with isolated mitral stenosis. Actually, in 50 of the last 62 patients operated upon, with a preoperative diagnosis of mitral stenosis alone, at our Clinic have been operated upon from the right side. The chief apparent advantages of the right-sided approach to the stenotic mitral valve may be summarized thus:

1. It is feasible routinely to explore the tricuspid valve by way of the right auricular appendage and to perform, if necessary, tricuspid commissurotomy for stenosis, or surgical plication[18] of the mural portion of the tricuspid annulus fibrosus for incompetence.

2. It is feasible routinely to palpate the external surface of the root of the aorta and the left ventricular outflow tract (and the septal leaflet of the mitral valve) in order to recognize the presence of aortic valvular disease, and directly to attack this lesion immediately, if deemed advisable. In certain of the patients, the aortic cusps may be examined digitally by passing the intracardiac finger through the mitral valve orifice and up to the aorta.

3. Ability routinely to explore the interatrial septum from the right (by way of the right auricular appendage) and to carry out efficiently and immediately an atrio-septo-pexy procedure if an interatrial septal defect is found. Previously unsuspected interatrial septal defects were found in 2 of the patients so explored during the course of a mitral commissurotomy from the right side and were promptly corrected.

4. Ability to approach the mitral valve without disturbing either fresh or old clotted material which is found in approximately 25 per cent[13, 38] of all patients operated upon for mitral stenosis. Since fragmentation of such clots is the chief source of operatively produced arterial embolization, a serious complication which has occurred in 6.1 per cent[13] of our entire series of mitral commissurotomy operations performed from the left-sided approach (1,500), it would seem that avoidance of this complication should be an operative objective of primary concern to the surgeon. Such clots extend well into the true atrial chamber in 13.6 per cent of those affected,[13] sometimes coming to occupy 25 to 30 per cent of its entire luminal capacity. Apparently (unlike fibromyxomatous

interatrial tumors) they never are found attached directly to the interatrial septum. Hence, a right-sided entrance into the left atrium by way of the interatrial groove would seem "always" to avoid disturbing such a thrombus.

5. Technically, a superior type of mitral commissurotomy may be accomplished from the right side. In large part, this is related to the relative ease and safety experienced in separating the posteromedial mitral commissure, a maneuver often fraught with difficulty and danger and hence usually deleted whenever possible by most operators, when mitral commissurotomy is carried out through the usual left-sided approach.

6. The presence of a nearly virgin operative field in a patient who previously has been subjected to commissurotomy from the left side. This problem will be encountered more frequently in the future as mitral commissurotomy is undertaken more often at an increasing number of clinics. Since, in most instances, the left auricular appendage will have been obliterated surgically, this advantageous "tunnel" of entrance[38] from the left usually will have been destroyed.

7. Extensive calcification of an old laminated thrombus lining the left lateral aspect of the left atrium (a relatively rare complication) in the presence of small pulmonary veins may require the use of the right-sided operative approach.

8. The supine operative position employed for the right-sided approach physiologically is easy on patients with borderline circulatory stability. Many of these patients cannot tolerate the lateral position which is preferred for the performance of mitral commissurotomy from the left side. They may develop severe hypotension as soon as they are turned into the lateral operative position.

9. In patients operated upon by the right-sided approach, obviously there is no danger of laceration of the left auricular appendage. This is of significance only in that the respective appendages are the thinnest, the most fragile, and, hence, the most easily lacerated portions of the entire cardiac wall.

10. The atrial attachment of a transvalvular pericardiocartilaginous "stent" used for the correction of mitral regurgitation of the posterior "teardrop" variety is best placed in the region of the right interatrial groove in order to ensure the persistent accurate application of the wider extremity of the cross-sectional contour of the stent into the rounded incompetent posterior "commissure."

11. In the presence of a persistent and recurrent right-sided pleural effusion (from heart failure), the right-sided approach permits one to avoid surgically collapsing the functional left lung, and at the same time to correct the pleuropulmonary condition by lung decortication.

12. Postoperatively, there is less incisional pain and delay in re-establishing arm function with the right-sided anterior thoracic wound.

13. A more esthetic operative scar and a less crippling operative incision result from the right-sided approach. Since the right anterior incision is placed in the inframammary groove in the female, it readily may be hidden by the usual brassière type of breast support (see Fig. 26, *B*). The patient, often a young woman, then may wear a low-backed or low-necked gown without psychologic stress. In the male, the crippling effect of splitting and surgical

division of a relatively few fibers of the right pectoral muscles is insignificant in comparison with the great muscle damage produced by interruption of the continuity of major portions of the rhomboideus, latissimus dorsi, serratus, and (sometimes) erector spinae muscle groups (see Fig. 26, *C*).

On the other hand, the disadvantages associated with the right-sided approach to the mitral valve seem to be:

1. Somewhat less than the usual facility will be experienced in separating the fused anterior mitral valve commissure from the right. This is related largely to an inability to apply appropriate external counter pressure over the commissure. Hence, an instrumental division of this commissure is required more frequently than when the left-sided route is used.
2. One experiences considerable difficulty in dissecting up the innominate and left common carotid arteries from the right-sided incision in an effort to diminish the incidence of cerebral embolization during the course of surgery. As mentioned, this disadvantage largely is nullified in our opinion by reason of the avoidance of direct manipulation of a possible thrombotic appendage when the valve is approached from the right.
3. One cannot plicate the mural portion of the fibrous ring of the mitral valve in order to correct mitral regurgitation of the annular type from the right side.
4. Obviously, the left auricular appendage cannot be ligated through a right-sided approach. Hence, one is unable to ensure that arterial embolization at a later date may not derive from thrombosis of the unobliterated left auricular appendage. We feel that adequate enlargement of the mitral orifice offers a reasonably reliable guarantee against further atrial thrombosis, and, hence, against arterial embolism as indicated by the study of Storer and associates.[46]
5. Inability to massage the ventricles properly through the right-sided incision in case of cardiac arrest. Such an accident would demand prompt opening of the left hemithorax and usually transection of the sternum.
6. There is always a possibility of losing hemostatic control of the atrial incision due to the unexpected detachment, breaking, or inadvertent cutting of the constricting purse-string suture placed about the dissected portion of the interatrial groove. Simple approximation of the two lips of the dissected interatrial groove with rat-toothed thumb forceps or Allis clamps will stop this type of bleeding.
7. A freely floating or "ball-type" of intra-atrial thrombus is more difficult to remove from the left atrium by the right-sided approach.
8. A surgeon with a short index finger may experience difficulty in separating the anterior mitral commissure in a patient with a very large left or dilated atrium.

When all is considered, we have come to the conclusion that since rheumatic valvulitis tends to present multiple sites rather than a single site of valvular involvement and since clinical and physiologic investigations in their present state of development are rather crude and often unreliable, it is better to explore digitally (only by external palpation of the aortic valve, however) and to treat definitively and immediately, if necessary, all three valves which may be subject to such involvement in nearly every case operated upon for any one such lesion. It is obvious in order to implement this concept that the right-sided approach should be chosen.

Today, stenotic lesions of both the aortic and mitral valves readily are correctible through an incision made in the right third intercostal space. Furthermore, the possible presence of tricuspid stenosis which unfortunately appears often to be undiagnosable by the usual methods of preoperative investigation, readily may be determined with certainty by direct digital palpation using the same thoracic incision.

We now feel that every case of acquired aortic stenosis, with or without evident mitral involvement, of a severity sufficient to justify surgery should be operated upon by way of an anterior right third intercostal incision using a transaortic approach to the valve. Since the right auricular appendage lies over the first portion of the aorta, it may tend to "get in the way" of the operator. Therefore, we prefer to explore the tricuspid valve digitally by way of the right auricular appendage before directing our efforts toward the aortic lesion. If tricuspid stenosis is found to coexist, it may be relieved either by digital pressure or by digitally guided instrumental means. Then, the appendage is amputated partially. It is ligated at its base, and is oversewn.

After the aortic commissurotomy has been completed, thought is given to the advisability of exploration of the mitral valve. If the clinical features of the case suggest combined valvular involvement (especially the presence of atrial fibrillation), or if the tricuspid valve has been found to be stenotic (apparently this is never found in the absence of coexistent mitral stenosis), the mitral valve should be explored digitally by way of the dissected interatrial groove. Indeed, if the patient's condition is not precarious after the completion of the aortic surgery, it is the authors' custom routinely to explore the mitral valve in order at least to rule out a possible unsuspected obstruction. If stenosis is found, an appropriate digital or instrumental mitral commissurotomy is carried out from the right side.

Coexisting mitral insufficiency, in a patient with aortic stenosis, is usually of a functional nature and relatively seldom requires definitive treatment. When it does, the procedure of applying a transvalvular pericardiocartilaginous stent as described elsewhere[38] may be undertaken.

Selection of Patients.—In general, it is felt, as in selecting patients for mitral commissurotomy, that all patients with aortic stenosis who might be considered as belonging in functional Class II or III (New York Heart Classification) may and should undergo aortic commissurotomy using the supravalvular approach. It has been our policy to operate upon Class IV patients whenever their condition can be brought to such a point that it is felt they logically might be expected to survive surgery. Whether this attitude will prove to be unduly optimistic, only time will tell.

In the past, we have leaned heavily upon the brachial arterial pressure curve in patients who presented somewhat questionable indications for surgery. While we fully realize the shortcomings of this method of evaluation, it does offer an additional and an essentially safe method of evaluating the functional importance of the aortic narrowing.

The ideal method of measuring the degree of existing aortic stenosis, of course, is left heart catheterization, and this study is now applied routinely in all of our patients who are suspected of having aortic stenosis This may be carried out either by *puncturing the left atrium* by the use of a long needle inserted through a bronchoscope[39] or by inserting one through the intact thoracic wall (the right eighth intercostal space)[40, 41] We have always used the latter method A polythene catheter is passed through the needle to enter the left atrium Then, an attempt is made to cause it to traverse the mitral valve to enter *the left ventricle* *In some cases* (15 of 50, or 30 per cent), it may even be passed upward and into the aorta If this latter maneuver proves to be impossible, a simultaneously obtained brachial arterial tracing will afford a reasonably comparable pressure curve for the determination of any gradient which may exist between the ventricle and the aorta (see *Fig 27, A and B*) *Whenever possible, left heart catheterization* should be performed simultaneously with a right heart catheterization Thus a complete physiologic study and the relation of the gradients to the cardiac output may be established

At the same time, if any exists, a left atrioventricular gradient indicative *of mitral stenosis may be detected* We *do not* advise *aortic commissurotomy* unless a transaortic gradient of 40 mm Hg or greater can be established

Unfortunately, this desirable scientific study is not feasible in all patients We have only been able preoperatively to pass the catheter from the left atrium through the mitral valve and into the left ventricle in 88 per cent of our studied patients Furthermore, because of the inherent risks associated with the method (possibility of intrapericardial hemorrhage, arrhythmia, pneumothorax, infection), it probably will never be applied generally in many clinics in the evaluation of these patients

In children with congenital stenosis of the aortic valve, surgery is advised because of the high mortality associated with conservative management (Downing[42]) Here the approach chosen usually will be the transventricular one because the small aortic lumen may not admit the finger

Contraindications—It is felt that most of the pertinent contraindications to surgery for aortic stenosis are the natural common sense ones Certainly, no patient should be subjected to aortic surgery if he is in a terminal or unprepared state Neither should he be operated upon when in a phase of acute rheumatic activity or during the course of active subacute bacterial endocarditis

Coexisting significant regurgitant lesions of the mitral or aortic valves are severe deterrents to aortic commissurotomy However, we frequently accept such patients on a calculated risk basis if we feel that at the same time we will be able definitively to alleviate the regurgitation, or if we feel that the patient will receive some net gain from correction of the *stenotic lesion alone*

The finding of a very large heart is suggestive of complete myocardial exhaustion, and implies an inability of the myocardium to respond favorably even after proper relief of the obstruction Nevertheless, some clinically dramatic results have been obtained in occasional selected patients with very large hearts

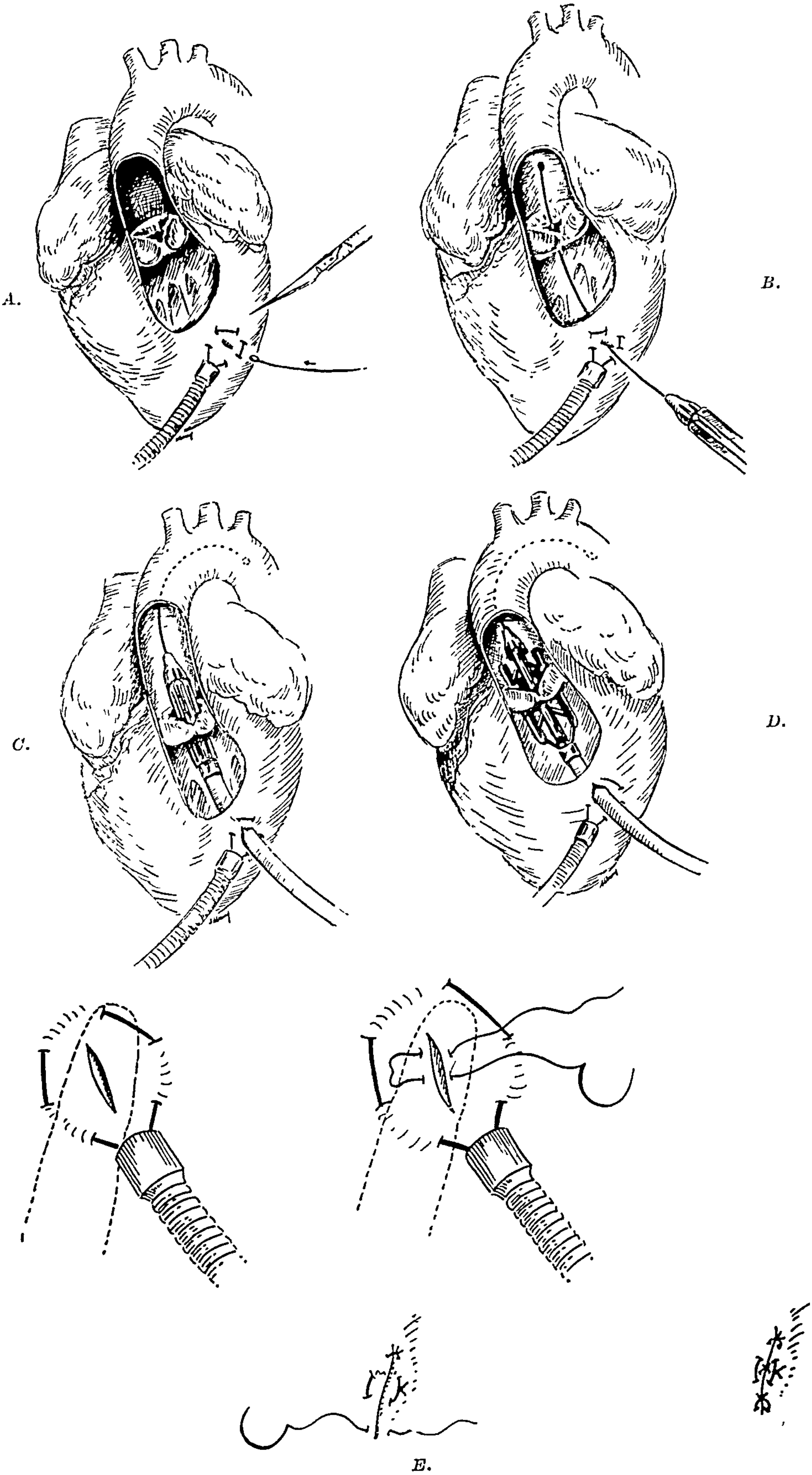


Fig. 18, A-E. *(For legend see opposite page.)*

to further advance of the instrument should be encountered at the valve level, the operator need not hesitate to force its entry since the instrument must follow the natural passage along the guide wire However, it is well first to draw the guide wire back until the olivary tip is palpable only 2 or 3 cm above the valve Otherwise there is no certainty that the increased resistance may not be the result of impingement of the olivary tip within a carotid or subclavian arterial orifice The lock on the handle which prevents the blades from opening is released Since the head previously will have been adjusted for free rotation upon the central shaft, its triangular cross section presumably will have become aligned automatically with the residual triangularity of the reduced aortic orifice Hence forceful compression of the handle grips should apply maximal parting or separating force against the central extremity of each line of commissural fusion The degree of possible expansion of the blades already will have been limited by a setscrew on the handle to the desired range (Fig 18, *A*, *B*, *C*, *D*)

Because of the possibility of "missing" the valve by inserting the dilator head too far, or not far enough, it is our practice to expand the blades at three different levels The first is the level that the operator feels is right, the next is placed 2 cm farther onward (toward the aorta) The last is made at a point thought to be 2 cm too far back toward the ventricle

The instrument is closed and is drawn smoothly out of the ventricle and the operator's left index finger is applied lightly over the ventricular puncture At least one mattress suture and three interrupted sutures are placed deeply to approximate the full thickness of the edges of the ventricular wound The needles are passed under the finger and the sutures are tied individually (Fig 18, *E*)

After hemostasis has been secured and vigorous heart action has been established, preparations are made to close the chest The cut pericardial edges may be closed partially, but a sufficient opening must be left to permit pericardial drainage into the left pleural space The pleura is drained by a No 26 F catheter with extra holes cut into it It is inserted through a stab wound made in the left seventh or eighth intercostal space at the posterior axillary line This is connected to a simple water seal drainage system

The chest wall is closed using approximating pericostal sutures of heavy chromicized catgut The muscles and fasciae may be closed with interrupted cotton or continuous chromic catgut We prefer to close the skin with subcuticular stainless steel wire

Fig 18—Technique of performance of aortic commissurotomy by the transventricular route *A* Tiny stab wound made in a selected relatively avascular area (near the left
ventricular apex cribe
wire is inserted left Extended guide
the interventricu rses ed upward along
becoming palpabl the olivary tip
and the closed upor nlarged slightly
instrumental hea aor one half of the
alignment of the cont el) Automatic
triangularity of the valve is assured by the vith the residual
head with the shaft *D* Parallel separation of junction of the
force against the central extremities of the maximal parting

E Method of digital surface tamponade of the left ventricular wound combined with moderate tension applied to the constricting purse string suture and the preferred technique of suturing the myocardium (From Bailey Surgery of the Heart courtesy Lea & Febiger)

Transaortic (Supravalvular) Digitally Controlled Aortic Commissurotomy.—

Incision: The patient is kept in the supine position after the induction of anesthesia and intubation. The right arm is brought to a 90-degree angle with the trunk at the shoulder. Although both sides of the anterior thorax and both axillae are prepared and draped, the incision is limited to the right hemithorax. In the female, the usual curved inframammary incision is selected and it is extended posteriorly up to the apex of the axilla. In the male, a more transversely directed incision, passing just below the nipple, is acceptable. In either sex, the anterior extremity should not lie farther cephalad than the level of the manubriosternal junction, and here should point nearly in a transverse direction. Then if sternal transection and a left-sided prolongation of the incision should be deemed advisable (perhaps to treat an element of coexisting or created aortic insufficiency), an esthetically curved extension of the wound will be possible.

The fibers of the pectoral muscles for the most part are split approximately in the line of skin incision being divided anteriorly for a limited extent. Bleeding points are picked up meticulously and are controlled either by ligation or by electrocoagulation. The third intercostal space is incised cautiously for entrance into the right pleural cavity. Division of the third costosternal junction affords an appreciable improvement in exposure especially in older individuals.

Great care is taken to dissect the internal mammary artery and vein separately. Both are divided between clamps and the four cut vascular ends are individually ligated with medium silk or cotton. A pair of fixed rib spreaders (Finochietto) is placed with the side bar toward the midline and the intercostal space is widely opened. Separation of the intercostal muscles posteriorly is extended by the blunt pressure of a gauze-covered thumb, facilitated considerably by the disruptive tension provided by the partially opened retractor.

Opening the pericardial sac: The right lung is compressed posteriorly, after dividing any restraining pleural adhesions if necessary. The right phrenic nerve is identified running longitudinally along the pericardium just anterior to the root of the lung. The pericardial sac is incised longitudinally about 1 cm. anterior to and parallel with it up to the level of the junction of the superior vena cava with the atrium, then deviating anteriorly to avoid injuring the former structure. Weighted traction sutures are placed in the posterior cut edge. The opposite extremity of the pericardial incision is directed forward at a right angle, turning at a level which is just caudad to the right atrial appendage. The anterior limit of this section passes under the pericardial fat pad and extends somewhat beyond the right sternal margin. This L-shaped incision results in the mobilization of an upper triangular segment of pericardium, pedicled obliquely forward and upward. Traction sutures placed in its free margins are attached with tension to the crossarm of the fixed rib spreader. This firm anterior pericardial suspension effectively acts to overcome any tendency of the mediastinal structures and the ascending aorta progressively to migrate toward the left. Such migration would render the exposure to the root of the aorta much less satisfactory.

The ascending portion of the aortic arch now will be visible within the wound, its first 2 or 3 cm. centimeters being overlapped by the right auricular appendage. Direct palpation of the root of the aorta, especially posteriorly, will reveal the typical systolic thrill of aortic stenosis, usually weakening or absence of the normal palpable shock of closure of the valve leaflets, and the diastolic thrill of associated aortic insufficiency if present. Tentative digital compression of the lowest portion of the aorta may reveal underlying masses of valvular calcification if marked. Sometimes a complete ring of calcification about the aortic annulus can be recognized.

Exploration of the right atrium: It is our practice in these patients first to explore the tricuspid valve by inserting the ungloved left index finger through an incision made in the tip of the right auricular appendage, hemostasis being ensured by a purse-string suture placed about its base. If tricuspid stenosis is found, it is treated immediately in a definitive manner using either the digital splitting or a digitally guided instrumental technique to separate one or more of the fused commissures. It is felt that an orifice sufficiently large to permit two fingers to pass simultaneously (4.5 sq. cm.) should be striven for, provided an opening of this size can be obtained without producing a serious degree of insufficiency. It is felt that the finding of a significant degree of acquired tricuspid stenosis, a lesion apparently never observed in the absence of a coexistent mitral stenotic lesion, renders it mandatory that the mitral valve be explored at this same operative session.

After examining or manipulating the tricuspid valve, the entire inner surface of the atrial chamber and septum is palpated to rule out the possible presence of a septal defect or of anomalous pulmonary venous drainage.

The exact steps of this procedure progress as follows: A noncrushing clamp, usually of the Satinsky type, is closed across the base of the appendage, usually encroaching somewhat upon the true atrial wall. Using a heavy silk or nylon suture with a curved Atraumatic needle, a circumferential purse string is placed about the excluded appendage toward its base. The ends of the suture are incorporated within a Rumel tourniquet. An incision is made in the tip of the excluded appendage, directed in parallel with the blades of the clamp. The lumen of the appendage is flushed with saline and any intervening trabeculae which might interfere with insertion of the finger are divided.

An assistant amputates the portion of the glove which covers the operator's left index finger and the latter is washed with saline and dipped into a dilute solution of heparin (1/1,000). The two lips of the appendage are held up by tissue forceps and the tip of the bare finger is inserted into the opening which first has been filled with saline. As the clamp is released, the finger is advanced into the atrial chamber and the tourniquet is tightened sufficiently to provide hemostasis.

The tricuspid valve is examined and it is determined whether its orifice is competent and of adequate size. If commissural fusion exists, it is evaluated. Then an attempt is made digitally to mobilize the adherent leaflets. If this fails with moderate pressure, one of the guillotine knives used for mitral commissurotomy is inserted through the appendageal incision along the palmar surface of the finger and one (or more) of the commissures is divided. In general we prefer to avoid anything other than relatively gentle digital manipulation of the anteromedial commissure, and to limit any necessary instrumental separation to the other two (Fig. 19, *A, B, C, D, E*).

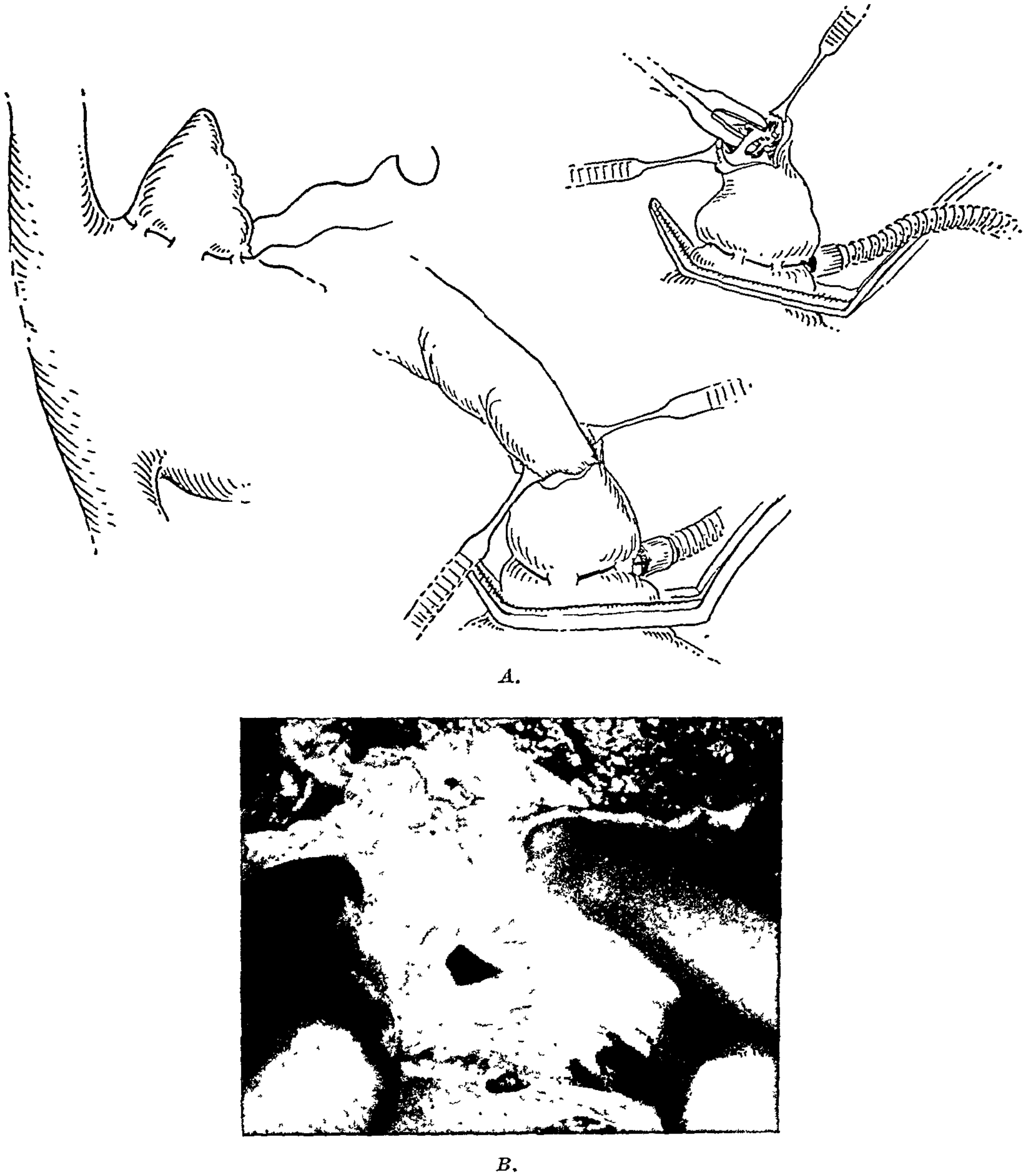
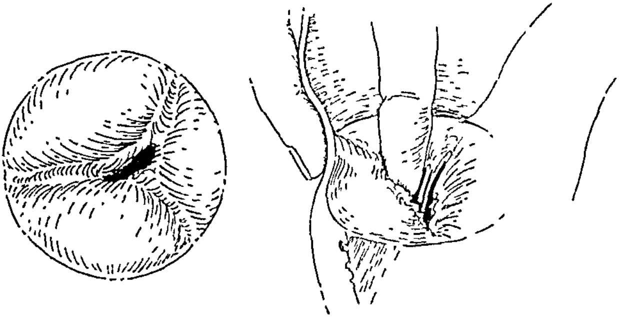


Fig. 19.—*A*, Steps in inserting the operator's ungloved left index finger into the right atrial chamber via the auricular appendage to explore or manipulate the tricuspid valve.

B, Symmetrial commissural obliteration in the tricuspid valve ultimately produces a diminutive central triangular orifice.

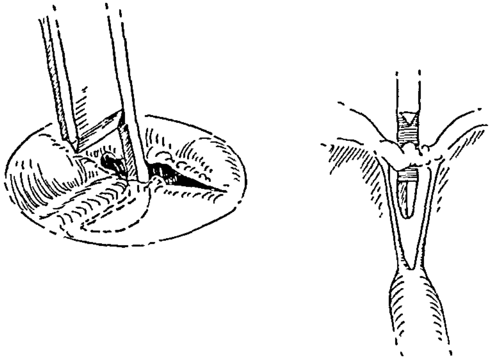
After opening the valve sufficiently, the instrument, if any has been used, is removed from the heart, and the septum and posterior portion of the atrium are explored. The fossa ovalis and the ostium of the coronary sinus routinely are included in this investigation. When the operator is satisfied with the examination, he withdraws his finger, reclamping the appendage.



C. D.

Fig. 19.—*C*, Asymmetric fusion of tricuspid valve commissures may lead to an eccentrically located or slitlike residual orifice.

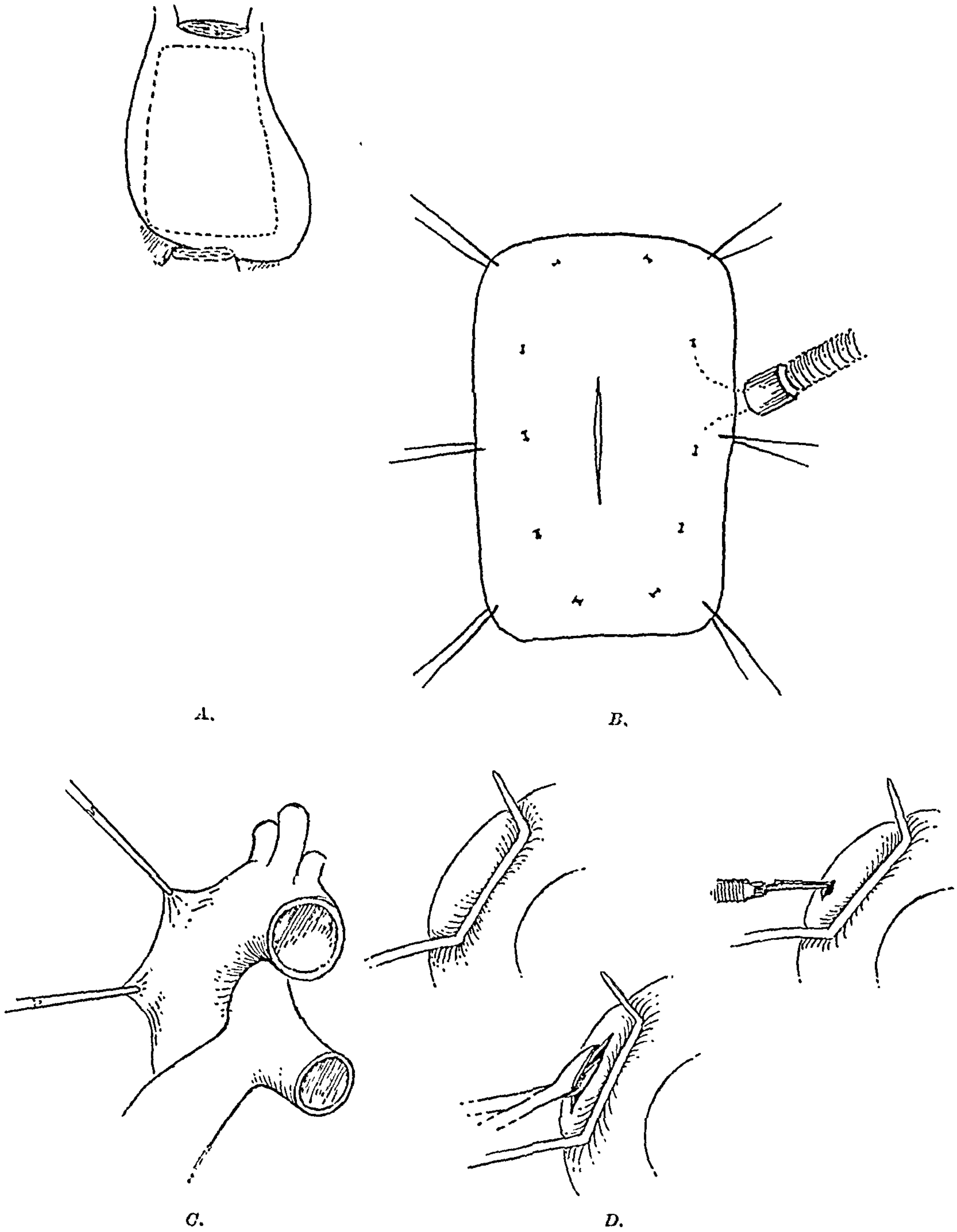
D, An attempt at digital splitting of the fused tricuspid commissures always is indicated before considering an instrumental attack.



E.

Fig. [illegible] of one of the usual mitral commissurotomy guillotines upon the [illegible] is essential if created insufficiency is to be avoided. The prope[illegible] between the two rows of chordae supporting that commissure a[illegible]he employment of a bare finger technique—because of its greater [illegible] essential in ensuring accuracy of placement of the instrument.

The purse string is tightened and tied as the clamp is removed, is passed again around the appendage, and is retied. The distal portion of the appendage is amputated to provide a biopsy specimen, and the cut end is oversewn with a No. 3-0 cotton suture on an Atraumatic needle. This reduces the size and lumen of the right auricular appendage greatly and exposes a significantly greater portion of the root of the aorta for the definitive surgery.



A. B.

C. D.

Fig. 20.—*A*, Resection of a large patch of the patient's own pericardium in order to create a pouch to be applied to the ascending aorta. This requires sternal transection and the opening of both pleural cavities. The employment of a preserved pericardial homograft enables the operator to limit the thoracic incision to the right third intercostal space. A patch of plastic fabric (Vinyon or sealed nylon) may be used in a similar fashion. *B*, A 4 cm. incision is made longitudinally in the patch of denuded pericardium or plastic fabric. A purse-string suture of heavy nylon thread is placed about its periphery in such a manner that the line of the suture loops comes to lie farther from the extremities of the incision than from the sides. Only very small loops of suture material are placed upon the smooth (epicardial) internal aspect of the pouch. *C*, The adventitia of the ascending aorta is picked up at selected points with fine hemostats. *D*, A large Satinsky type clamp is applied longitudinally to this portion of the aorta in such a manner as to exclude a sizable portion of its circumference (up to one-half). The excluded portion is incised longitudinally for a distance of 4 cm.

A segment of homograft pericardium obtained, sterilized, and preserved by the method reported by Meeker and Gross[30] for aortic homografts, is thawed out and any excess pericardial fat is dissected away. A rectangular patch 12 by 18 cm. is ideal for this purpose, although a smaller one (above 5 by 8 cm., however) may be used with somewhat more difficulty. While a portion of the patient's own pericardium may be used, this ordinarily necessitates opening both pleural cavities. If the right side of the pericardium is excised for this purpose, it must be replaced by plastic or other type of material to obviate possible postoperative dislocation of the heart into the right pleural space. While we and others, Glenn,[31] Swann[35] have used latex and plastic pouches instead of pericardium, their use often is attended by very serious bleeding from the aortic anastomosis. We feel that the use of a square patch of Vinyon plastic fabric or of nylon fabric in which the central and peripheral cut edges have been flashed in the flame of a match is much to be preferred. The technique is similar to that used for the pericardial patch.

The homograft is spread out upon a moist towel, epicardial surface uppermost. An incision, 4 cm. in length, is made longitudinally in the central portion of the graft. Using a heavy nylon suture with a swedged-on curved needle, the opening in the pericardium is circumscribed at a distance of 2 to 5 cm. from its edges. The bites of the needle are placed in such a manner that very small loops (2 to 3 mm.) of the suture material are placed on the epicardial surface of the membrane, while 1 cm. loops lie upon the roughened pleural surface of this tissue. The extremities of the sutures are caused to emerge from the rough surface. An effort is made to provide a greater margin of distance about the extremities of the opening than at its mid-point. This provision of additional tissue at these points tends to reduce the tension upon the aortic anastomotic sutures at the extremities of the suture line. Several weighted stay sutures are placed about the periphery of the patch to permit it to be held extended.

The adventitia of the right lateral aspect of the ascending aorta is picked up with fine hemostats, and a large Satinsky aortic clamp is placed longitudinally to exclude one-fourth to one-third of the aortic lumen at a site extending upward from a level about 2 cm. above the aorticoventricular junction. A 4 cm. incision is made in the excluded portion of the aortic wall and the lumen is flushed with heparin solution.

Now the pericardial patch is laid over the ascending aorta so that its incision lies upon the aortic opening and the free extremities of the purse string are placed medially. A special mattress suture of No. 4-0 arterial silk is placed at either extremity of the aortic incision in such a way that the angle of junction of the two cut pericardial edges is folded over, and then under the extremity in an imbricating fashion. These sutures are each continued as a running mattress, respectively, along one of the aortic lips, bringing about similar imbrication of pericardium over aortic edge for the full distance of the wound (Fig. 21, *A*, *B*). An extra mattress suture is placed through pericardium and aortic wall just distal to each of the initially placed ones in order to afford additional hemostatic security.

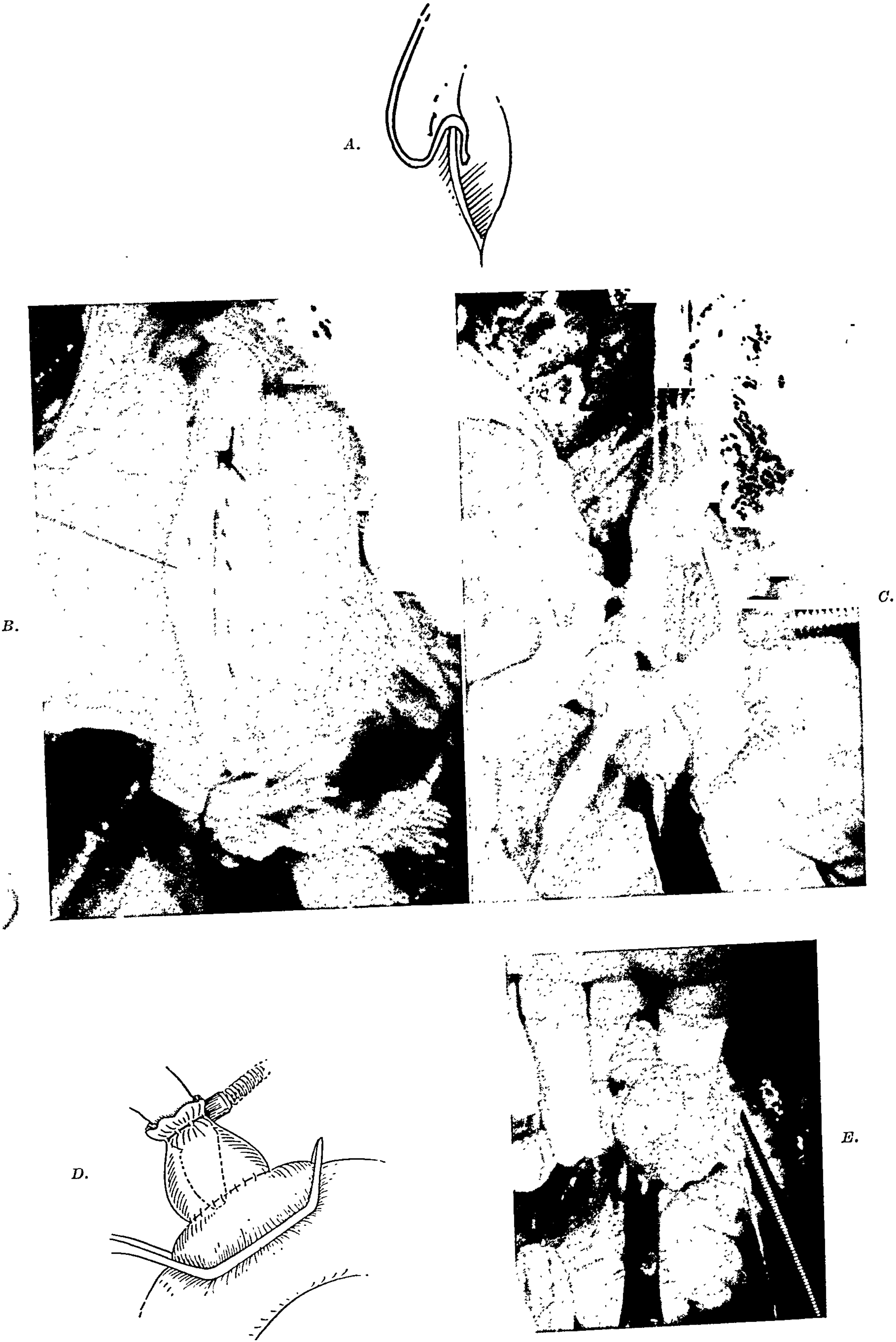


Fig. 21, *A-E.* (*For legend see opposite page.*)

Having completed the attachment of the pericardial patch to the aorta, the purse string is tightened by hand sufficiently to convert it into a "pouch-like" structure arising from the ascending aorta Then the extremities of the purse string suture are incorporated within a Rumel tourniquet The pouch is filled with dilute heparin solution and the tip of the operator's ungloved left index finger is inserted into it The purse string suture is tightened about the finger and the aortic clamp is released The pouch becomes distended with blood and pulsates but only insignificant bleeding should occur either from the line of anastomosis or about the entrance of the finger into the pouch (Fig 21, *C*, *D*, *E*) If such bleeding should be marked, the aorta must be reclamped and the leak repaired before going ahead with the definitive surgery

The operator advances his left index finger into the aorta as the tourniquet is loosened slightly Soon the finger tip approaches the valve and detects the jet of blood emerging from the ventricle, with each systole By following up the systolic jet, the finger tip will soon reach the diminutive valve orifice and be able to palpate it momentarily This may be repeated from time to time being careful never to block it for more than 3 heartbeats at a time, and allowing a somewhat longer interval of free circulation between such examinations Sometimes the valve opening may be so small that it can be recognized only with the greatest difficulty

An attempt is made to recognize the various commissures, and the concave upper surfaces of the cusps, and the sinuses of Valsalva are explored Having estimated the likelihood of restoration of some valvular function the finger tip is inserted into the diminutive opening, splitting it somewhat if necessary, and direct digital pressure is made first against that commissure which is considered most favorable for separation Within less than 3 heartbeats the pressure is released and the finger is drawn out of the valve orifice into the aorta for a few moments If splitting of this commissure has taken place and been adequate, attention is turned toward another one If separation has not begun, or has been incomplete, another attempt is made to split it digitally, this time with the aid of external counter pressure, applied by the gauze covered fingers of the right hand Sometimes hooking the intra aortic finger tip beneath the valve commissure and applying upward traction will prove a more effective way of breaking the cross fusion of the cusps However, this is a somewhat dangerous maneuver and may lead actually to partial detachment of one of the leaflets from the annulus fibrosus with resultant ensuing regurgitation (Fig 22, *A*, *B*)

It is felt that if one commissure has been split to the aortic wall, and another one half way, a minimally adequate valve passageway will have been established Nevertheless, the functional result may not be satisfactory if the

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Fig 21 —*A* Method of folding cut edge of central incision in pericardial or plastic fabric patch over lip of aortic incision Continuous through and through mattress sutures produce a permanently imbricated or mortised juncture of the tissues Such anastomoses do not leak even under the strain of full aortic pressure *B*, Actual photograph showing implementation of this concept *C*, By drawing upon the purse string a pouch without a lateral seam is formed *D* Insertion of ungloved left index finger of operator into pouch after filling it with saline solution Purse string tension is increased to prevent blood loss when aortic clamp is released *E* Actual photograph showing bare finger tip in pouch just before removing aortic clamp (From Bailey Surgery of the Heart courtesy Lea & Febiger)

valve components are relatively stiff and nonmobile. However, the mere fact of calcification need not necessarily indicate an incapacity of the valve to function. Provided there is significant flexibility in the basal portion of the valve or in the aortic wall itself at the site of valvular attachment, a considerable amount of normal type cusp movement may be established. In all cases as much anatomic valvular mobilization should be accomplished as is readily possible.

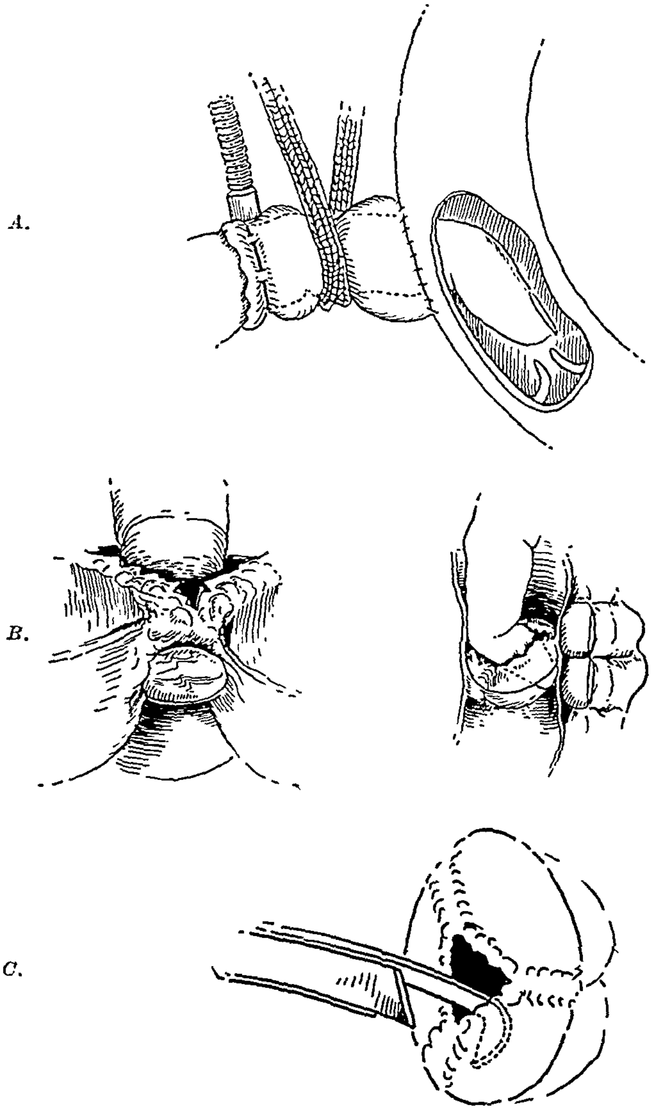

Fig. 22.—*A*, Finger tip is advanced into the aortic lumen and finally to the aortic valve level to explore its pathologic condition. A second line of hemostasis may be established by tensing a strip of gauze placed around the pouch proximal to the purse-string suture. *B*, In nearly half of the patients, one or more of the fused commissures can be separated by digital pressure, perhaps aided by external counterpressure with the opposite (right) hand. *C*, One of the mitral commissure guillotine knives may be inserted along the finger and through the pouch to divide a resistant commissure. Extreme accuracy of application is essential. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

Should the digitally established valvular enlargement be deemed to be inadequate or negligible, instrumental help becomes mandatory. In some cases one of the usual mitral commissurotomy guillotines may be inserted through the appendage along the finger. This technique was first applied by Swann.[35] It is passed through the valve orifice, is applied against an appropriate commissure, and is opened. The hooked underblade is engaged accurately under the line of valvular fusion, and then the instrument is closed by advancing the sliding guillotine-type blade. If accurately performed, a good anatomic separation of the valve elements may be achieved. However, the least inaccuracy of application may create a severe degree of aortic insufficiency (Fig. 22, *C*).

In many cases one may prefer to insert a two or three bladed retrograde aortic dilator through the opening in the pouch along the palmar surface of the operator's index finger. This may be accomplished without encountering excessive hemorrhage by first applying a constricting loop of unrolled gauze about the basal portion of the pouch. This permits relaxation of the distal purse string so that the lead end of the closed dilator may be placed within the opened upper portion of the pouch. The purse string is tightened about the instrument and finger, and the gauze is loosened slightly (but not completely, as it aids in the hemostasis). The instrument is advanced cautiously until its olivary tip lies just above the aortic valve. The intra-aortic finger locates the valve orifice (by the emerging systolic jet) and guides the tip of the closed instrument through it.

This technique may prove unusually difficult in some of the "clamshell" valves where there is an almost unrecognizably narrow slit which can be detected only by palpating the emerging systolic jetlike stream of blood. Here, the finger tip is poised on the valve edge in the region of the maximal jet. By timing with the cardiac cycle the flat edge of the finger tip may be inserted (perhaps forcibly) into the maximally open valve slit at the exact moment of beginning systole (beginning onset of the jet). To date this maneuver has been possible (and successful) in every patient operated upon by the supravalvular approach.

Once the finger tip has been inserted into a very small valve orifice (and has dilated it somewhat) it only requires exact timing to replace it by the tip of the closed instrument as the finger tip is withdrawn. Deeper insertion to apply the shoulder and dilating portion of the instrument into the valve may entail the use of considerable force.

The finger and dilator are turned in such a way as to line up one of the parting blades against the commissure deemed most favorable for blunt disruption. The other blade automatically becomes lined up with another commissure. By cautious compression of the hand grips of the instrument, the two blades apply pressure against the appropriate commissures, nearly always splitting at least one of them. Any intimation that one blade has begun to make pressure directly against the aortic wall or that valvular separation has begun at a point other than at a commissure is an indication for re-evaluation of the problem or termination of the procedure. By rotating the instrument 120 degrees, the third commissure may be attacked. After use of the instrument further digital splitting may become possible, just as it often is in the

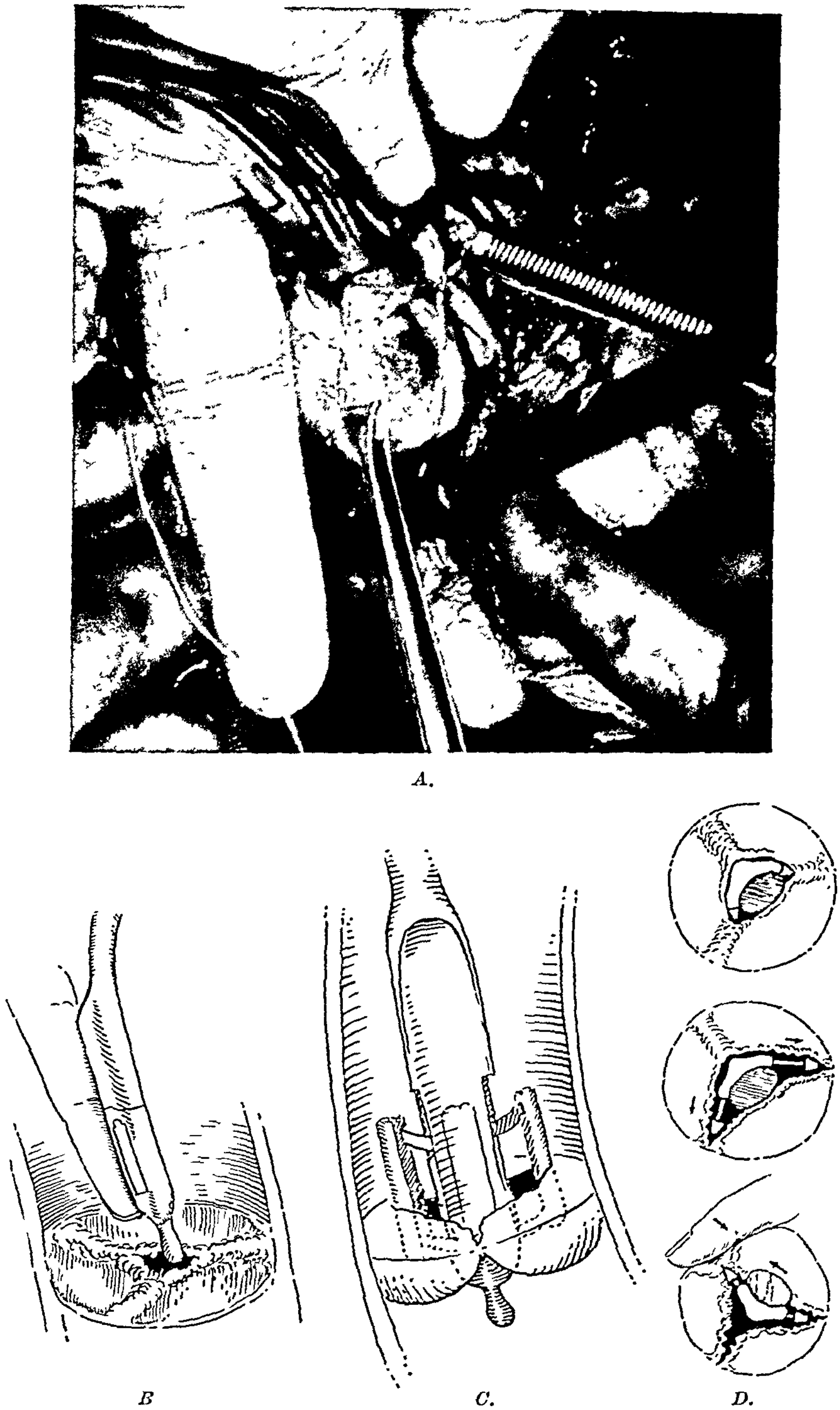

Fig. 23.—*A*, Two-bladed retrograde dilator, illustrated in Fig. 17, is inserted through the pouch along the palmar surface of intra-aortic finger to separate resistant valve commissures. *B*, Olivary tip of dilator is guided by intra-arterial finger to the diminutive valve orifice by the technique of following up the pulsatile jet of blood emerging from the nozzle-like valve opening. *C*, After the dilator mechanism has been forced into the valve orifice and has become aligned with two of the commissures, the blades are caused to expand by compressing the handles of the dilator, thus applying splitting pressure simultaneously against two of the fused valve commissures. *D*, Representation of method of applying parting pressure against each of the three valve commissures. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

course of a digitally guided mitral commissurotomy. Indeed the whole procedure closely resembles that of the now well established comparable surgery of the stenotic mitral valve (Fig 23, *A, B, C, D*).

Upon the termination of valvular instrumentation, the operator reassures himself (by intra-arterial examination) that the blades of the dilator are completely retracted (closed) and then simply withdraws the instrument from the vessel and pouch. Any attempt at withdrawal with the blades even slightly expanded is fraught with great danger of engagement upon a portion of the valve or of laceration of the aortic wall itself.

The finger tip is withdrawn into the pericardial pouch and a generous portion of the aortic wall, beyond the basal attachment, is clamped with the special Satinsky clamp. The attaching sutures at the base of the pouch are cut away and the aortic incision is repaired using one row of continuous mattress sutures of No. 4-0 arterial silk, and one more superficially placed row of continuous over and over sutures. After releasing the clamp, simple digital pressure with a gauze pad will suffice to control any oozing from the stitch holes.

Exploration of the mitral valve. Providing the patient's condition is satisfactory after the completion of the closure of the aortic wall, it is felt that a digital exploration of the mitral valve is indicated routinely. All too often in spite of the exhibition of all the usual clinical and physiologic methods of investigation, a dynamically significant mitral valvular lesion may be overlooked in a patient with preponderant aortic disease. Should the operator fail simultaneously to correct such a lesion, especially if it is a stenotic one, significant over all clinical improvement may not occur, since the cardiac output will not increase appreciably as a result of relief of the aortic obstruction alone. Hence, since mitral stenosis is present in about one half of all patients operated for aortic stenosis, we feel that such a coexistent lesion should be ruled out in all cases, if the patient's condition permits, by direct digital examination of the mitral valve. Naturally, if a strong suspicion of coexisting mitral stenosis has been expressed by some of the medical attendants, one's desire to rule it out definitively is enhanced. The presence of atrial fibrillation usually indicates associated mitral disease since it rarely appears in uncomplicated aortic valvular disease but is very common with mitral lesions. The previous finding of a serious degree of tricuspid stenosis is considered to be a pathognomonic indication of the presence of mitral stenosis.

The interatrial groove is exposed just to the right of, and posterior to, the superior vena cava. It is dissected apart for a distance of about 1.5 cm. Then a small bite purse string suture is placed widely (5 to 6 mm. distance) about this area of dissection, using a curved needle swedged on a heavy nylon suture. Care is taken that each pass of the needle penetrates into one of the atrial lumina. The ends of the suture are incorporated within a Rumel *tourniquet* for reliable flexible hemostatic control (Fig 24, *A, B, C, D*). A No. 3-0 cotton suture is placed and tied at each extremity of the dissection to facilitate subsequent reparative suturing.

The interatrial septum is dissected somewhat farther, perhaps to a depth of 2 cm. taking great care not to break, inadvertently, into the right atrium. The ungloved tip of the operator's left index finger is inserted into this dissected area. A large knife blade (Bard-Parker No. 20) on a detachable handle is laid upon the palmar surface of this bare finger and is advanced over the tip to penetrate through the left or lower half of the septum and to enter the chamber of the left atrium. The knife is withdrawn with care to avoid cutting the purse-string suture which is kept loosened at this time. The finger tip seeks out the site of hemiseptal puncture, tamponading it and then advancing through it to the region of the mitral valve. This valve lies farther from the right-sided site of cardiac entrance than it does when approached through the more usual left-sided incision, and, therefore, may necessitate the insertion of nearly the whole length of the finger into the atrium (Fig. 25, *A, B, C, D*).

The valve slit runs approximately horizontally with the patient in the supine position, and, hence, nearly in line with the finger. This is at right angles to its general line of direction when examined from the left lateral approach. After exploring and evaluating the anatomic and functional condition of the valve, thought is given to the technique of performing a commisurotomy if mitral stenosis has been found or some type of repair if the valve is incompetent. The latter is a more major undertaking and will not be detailed in this paper. However, the former is so readily and logically accomplished from this approach that it will be given at this time.

Mitral commissurotomy from the right side: The tip of the bare index finger is directed first against the least accessible (anterolateral) commissure of the valve. In a fair percentage of cases (20 to 30 per cent) anatomic splitting will take place even without the exhibition of much pressure. However, really strong disruptive pressure against this commissure can scarcely be applied from this approach since effective external digital counterpressure is well nigh impossible. Hence, the use of an appropriate instrument may become necessary, unless the posterior commissure can be opened sufficiently by itself to provide an adequate final passageway (1½ to 2 fingers or 3.0 to 4.5 sq. cm. cross-sectional area).

Digital separation of the very readily accessible posterior commissure is accomplished much more easily from this approach than from the more usual left lateral one through the left auricular appendage. As in the digital performance of commissurotomy, by any approach, it must be done only a little at a time by intermittent application of pressure, in order to avoid a sudden great extension of the separation which might be complicated by severe regurgitation. Probably the posterior commissure can be split digitally to some degree in at least 50 per cent of patients thus operated upon.

When an incompletely satisfactory opening is obtained by these maneuvers, instrumental aid becomes mandatory. Our preferred instruments are essentially longer versions of the usual commissurotomy guillotines* modified as suggested by Nichols[43] so that they may be used also to divide a directly adherent papillary muscle should it prove necessary.

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*Obtainable from George P. Pilling & Son Co., Philadelphia, Pa.

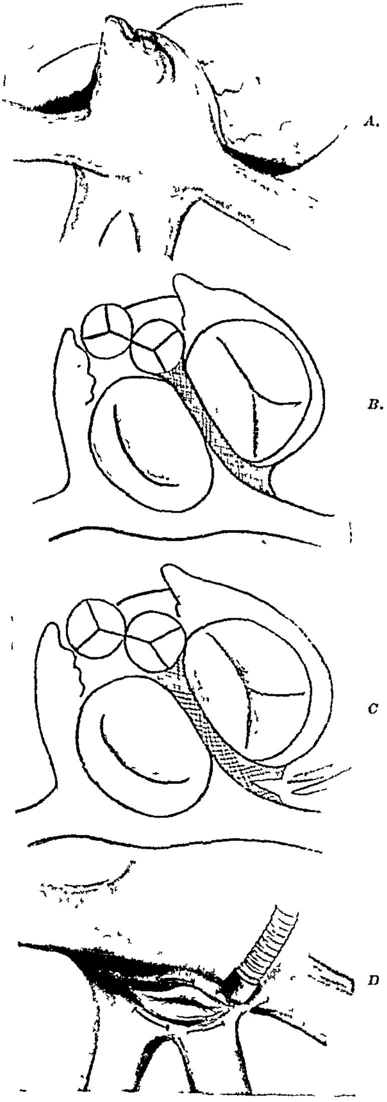


Fig. 24.—*A*, Pertinent anatomy concerned with the performance of mitral commissurotomy from the right side, via the interatrial groove *B*, Cross section of the [illegible] vel of the four [illegible] diagonal line [illegible] ptum which is [illegible] to two halves [illegible] the interatrial groove and septum *D*, Not over 1.5 cm. of the length of the interatrial groove is separated A purse-string suture of heavy nylon thread is placed about this area of dissection, taking care that each bite penetrates into the respective atrial lumen A Rumel tourniquet is employed to provide flexible hemostatic tension.

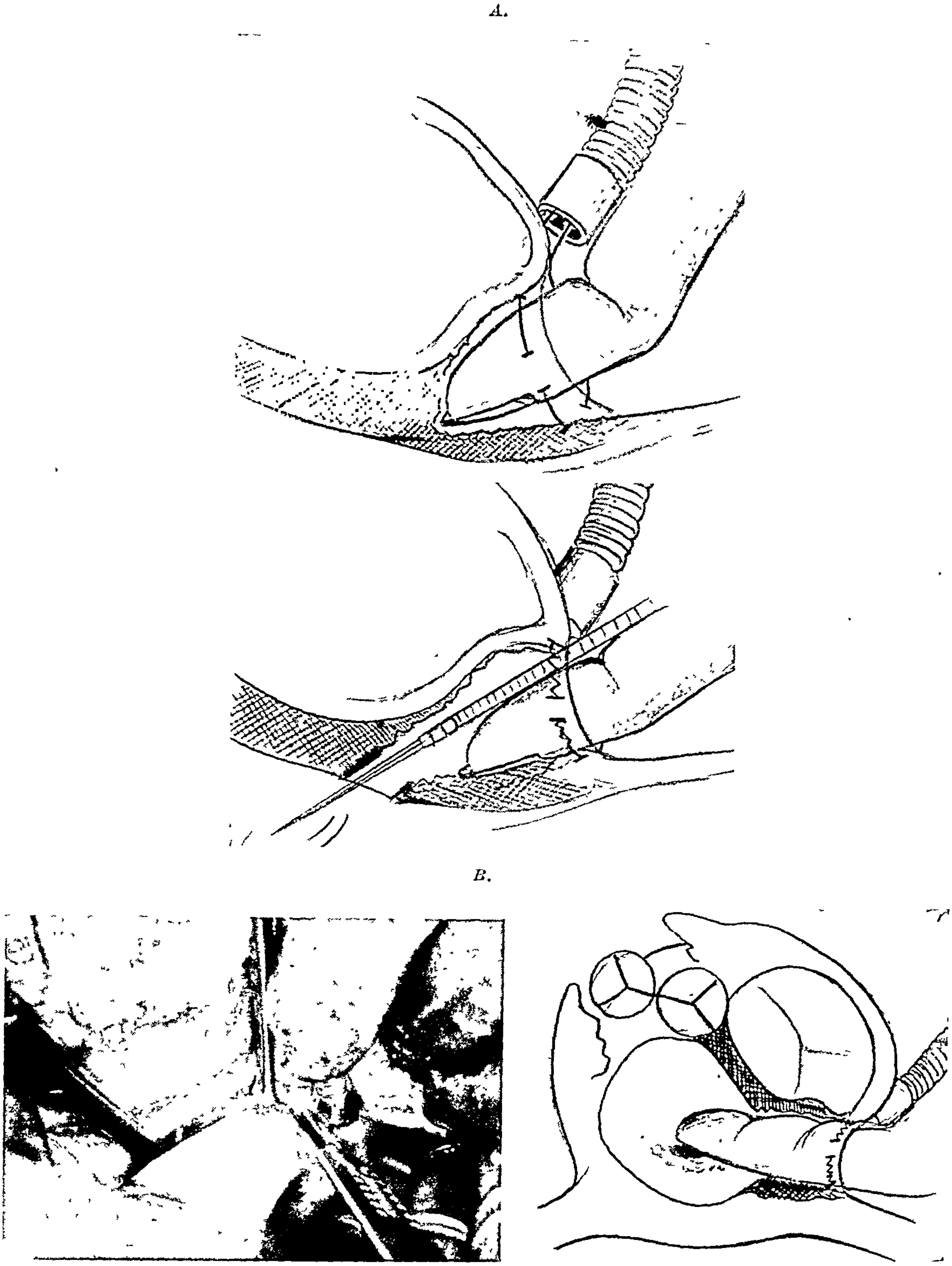


Fig. 25.—*A*, The dissection of the interatrial septum is carried to a depth of 1.5 cm., in part by digital separation.

B, A large-bladed knife (Bard-Parker No. 20) is laid upon the palmar surface of the bare finger tip as it lies in the furrow of dissection. The direction of insertion of the knife is angulated and it is advanced over the tip of the finger until it pierces the left side of the separated septum. It should not penetrate farther than 1 cm. within the left atrial chamber lest it pierce the posterior (opposite) atrial wall.

C, Actual photograph of knife puncturing into left atrial chamber via the interatrial groove. Care must be taken to avoid dividing the encircling purse-string suture which is kept loose during this maneuver. A gush of bright red blood indicates entry into the left atrium.

D, As knife is withdrawn, the operator's finger tip is advanced, seeking and finding the site of puncture of the left half of the atrial septum and penetrating through it The septal opening will be found to be somewhat too small for the finger, thus providing a secondary hemostatic line of constriction.

For the *anterior commissure* the left-sided guillotine is inserted into the heart along the bare palmar surface of the intracardiac finger as the anterior lip of the dissected sulcus is held upward with hand-held tissue forceps. It is passed through the valve orifice in the closed position and is applied against the anterior (less accessible) commissure taking care that it comes to lie between the two rows of chordae, respectively, supporting the two leaflets. These lie more posteriorly (toward the vertebrae) than would be expected. The sliding blade is retracted by the right hand while the intracardiac finger tip presses the edge of the instrument against the extremity of the valve orifice. As the sliding blade is retracted the hooked underblade slips beneath the commissure. At this point the handle of the instrument is maneuvered so that the cutting blades are directed horizontally. Pressure of the right thumb closes the instrument, the sliding blade shearing through 4 mm. of the thickened commissural tissue. If the tissue is deemed thick and fibrous enough to justify a second cut the sliding blade is drawn back without removing the instrument from the valve, permitting the hooked underblade to pass further under the commissural tissue. Maintenance of finger tip pressure against the cutting extremity of the instrument ensures exact placement of the instrument into the apex of the previous incision.

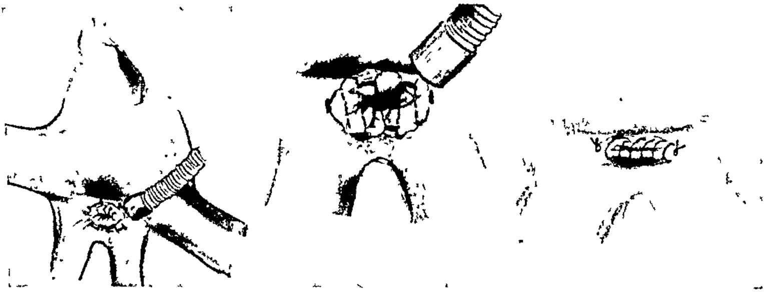
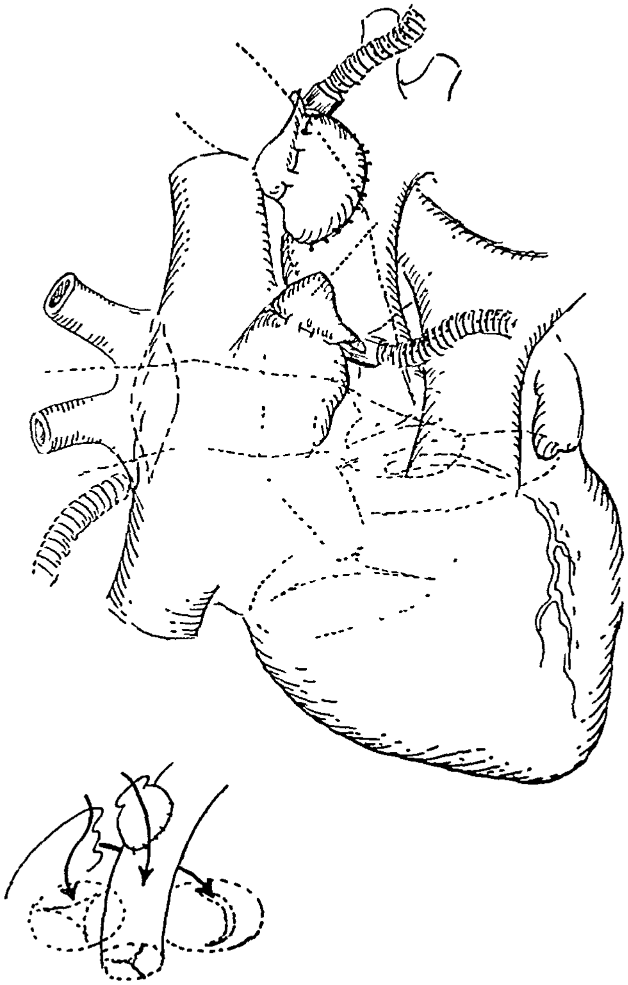


Fig. 25.—*E*, Steps in [illegible] portion of the interatrial septum after the finger is withdrawn. [illegible] initial hemostasis. Application of a row of continuous mattress [illegible] dissected lips of the interatrial groove. Final oversewing stitches reinforce closure No attempt is made directly to suture the edges of the incised left half of the septum. Purse string may produce puckering of inner atrial wall and should be removed.

Often the operator will prefer to remove the instrument from the valve momentarily, while he applies tentative disruptive pressure to the partially incised commissure. If separation then begins with digital pressure, the instrument is removed from the heart and the commissurotomy is completed by a splitting technique.

If an inadequate valve opening has resulted from these efforts, instrumentation of the posterior commissure is in order. This is not attended by the same considerable risk of production of insufficiency which has been experienced by nearly all operators in attempts at posterior commissural instrumentation from the left side. The right-sided guillotine is inserted the left atrium in the manner used previously for the left-sided one. It is passed into the valve orifice in a closed position and is opened while its edge is being pressed back against the posterior commissure. Again great care must be taken to apply it so that the hooked underblade becomes placed accurately between the two rows of supporting chordae. This commissure is cut in the same manner as is the anterior, and the commissurotomy is continued digitally if and when this method of leaflet mobilization becomes effective.



A.

FIG. 26, *A*.—Sketch showing how all three of the valves which are subject to clinical rheumatic involvement may be approached from a right-sided thoracic incision for exploration and corrective surgery. The aortic valve may be approached through a pouch sutured on the ascending aorta, the mitral through the dissected interatrial groove, and the tricuspid through the incised right auricular appendage.

Subvalvular cross fusion of the chordae or of the papillary muscles themselves should be handled in exactly the same manner as they are through the left-sided approach to this valve. Nichols[44] has described these methods in some detail. Since the guillotines designed especially for the right-sided approach have been made to include the principle of his Papillotome, no additional instrument of that type is necessary.

In general, when instrumentally dividing a commissure from the right, *one must remember* that, in this position, the extremities of the deformed commissures are not usually directed above the horizontal. Hence, the line of the cutting blades of the instrument, when dividing either commissure, ordinarily should be directed horizontally rather than upward into the substance of the preponderantly important septal leaflet. However, the line of valve fusion then turns upward at either extremity so that a second cut should be deviated in that direction.

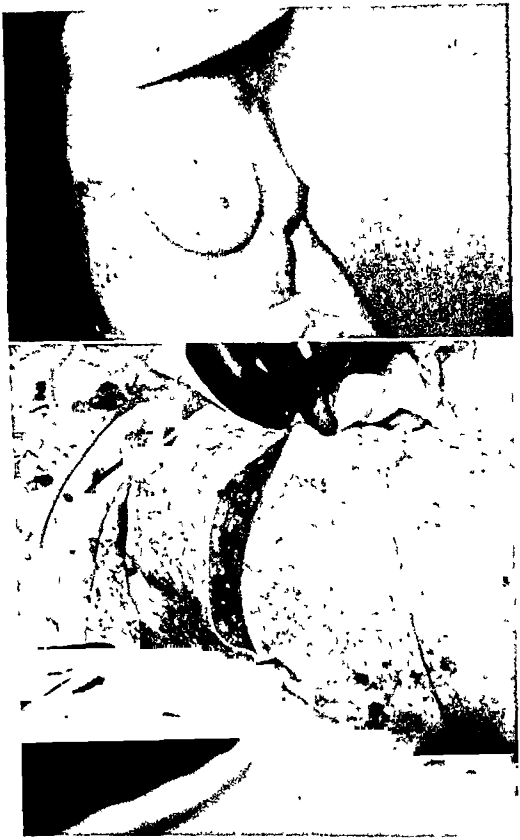


Fig. 26, *B* and *C* — *B*, Photograph of right-sided incisional scar in patient subjected to simultaneous corrective surgery of all three valves *C*, Photograph of the less esthetic posterolateral thoracic incision employed in the usual left-sided approach to the mitral valve

When the definitive mitral surgery has been completed, the finger is withdrawn gradually from the heart, the purse-string suture being tightened progressively. When the finger has been removed entirely, the puckered anuslike opening between the atria is sutured together with cotton (3-0), using one row of continuous mattress sutures and one row of simple running sutures. No attempt is made to suture the slitlike opening which has been created in the left side of the interatrial septum (Fig. 25, *A, B, C, D, E,* and Fig. 26, *A*). The tourniquet and the purse string are removed.

The pericardium is flushed with saline and is repaired loosely with interrupted cotton sutures. The pleura is flushed and is drained with a No. 26 F. urethral catheter with additional holes cut into it. This is passed through a stab wound in the right eighth intercostal space, posterior axillary line, and is connected with a water-seal drainage system.

The ribs are approximated with heavy pericostal sutures of chromicized catgut. The muscles and fasciae of the chest wall are closed in a similar manner. The skin is closed with continuous subcuticular stainless steel wire. This incision produces a much shorter and cosmetically more acceptable scar than does the older left posterolateral one (Fig. 26, *B, C*).

RESULTS OF AORTIC COMMISSUROTOMY

Naturally, it is impossible to present the desirable long term follow-up in a series of patients subjected to aortic commissurotomy since the first of them was operated upon (by a reasonably acceptable technique) only three years ago (April 4, 1952). Furthermore, the actual clinical and statistical results obtained in a beginning series in a pioneering effort directed toward the alleviation of a previously inoperable lesion should be evaluated both generously and realistically. The nearly continuous changes in methods of case selection, in surgical technique, and in medical management have vitiated many possibilities of nice and meticulous comparison. The coexistence of other valvular conditions in many of the patients has added other variables to our consideration. In retrospect, we can see that there have been errors in concept, in judgment, and in technique. The knowledge which now permits their recognition and correction, however, could scarcely have been obtained other than by the frequently painful one of trial and error.

A total of 287 patients with aortic stenosis have been operated upon since March 6, 1950. All but those with essentially "pure" aortic stenosis and combined aortic and mitral stenosis operated upon by the triradiate transventricular dilator or through the supravalvular approach have been eliminated from further critical evaluation. Twenty-one and three-tenths per cent of the patients were operated upon by the much preferred supravalvular or transaortic approach using an artificial "aortic appendage" and a digital or a digitally controlled technique. The operative mortality and the postoperative results obtained with the transventricular operations appear on Tables I, II, IV, and V.

TABLE IV. RESULTS OF 68 PATIENTS OPERATED UPON FOR AORTIC STENOSIS BY THE TRANSVENTRICULAR APPROACH. ONE- TO THREE-YEAR FOLLOW-UP

AMERICAN HEART ASSOC. CLASS	NUMBER OF CASES	OPERATIVE DEATHS	LATE DEATHS	SURVIVORS					LOST TO FOLLOW-UP
				MARKEDLY IMPROVED	IMPROVED	SAME	WORSE	PERCENTAGE IMPROVED OR MARKEDLY IMPROVED (%)	
I	3	1 (33.3%)	-	-	2 (100%)	-	-	100	
II	19	2 (10.5%)	2 (10.5%)	3 (23%)	8 (61.5%)	1 (7.7%)	1 (7.7%)	81.5	2
III	41	16 (36.4%)	8 (18.3%)	4 (21%)	12 (63.2%)	1 (5.3%)	2 (10.5%)	81.3	1
IV	2	0	2 (100%)	-	-	-	-		
Total	68	19 (28%)	12 (17.7%)	7 (20.6%)	22 (64.7%)	2 (5.9%)	3 (8.8%)	85.3	3

TABLE V. RESULTS OF 87 PATIENTS OPERATED UPON FOR MITRAL AND AORTIC STENOSIS BY THE LEFT-SIDED APPROACH. SIX-MONTH TO THREE-YEAR FOLLOW-UP

AMERICAN HEART ASSOC. CLASS	NUMBER OF CASES	OPERATIVE DEATHS	LATE DEATHS	SURVIVORS					LOST TO FOLLOW-UP
				MARKEDLY IMPROVED	IMPROVED	SAME	WORSE	PERCENTAGE IMPROVED OR MARKEDLY IMPROVED (%)	
I	0	-	-	-	-	-	-	-	-
II	14	-	1 (7.2%)	2 (15.4%)	7 (53.8%)	2 (15.4%)	2 (15.4%)	69	-
III	66	14 (21.2%)	3 (4.5%)	19 (39.6%)	25 (52.1%)	1 (2.1%)	3 (6.2%)	92	1
IV	7	2 (27.6%)	-	3 (60%)	2 (40%)	-	-	100	-
Total	87	16 (18.4%)	4 (4.6%)	24 (36.4%)	34 (51.5%)	3 (4.5%)	5 (7.6%)	88	1

TABLE VI. RESULTS OF SURGERY UPON 47 PATIENTS OPERATED UPON FOR AORTIC STENOSIS BY THE TRANSAORTIC APPROACH

AMERICAN HEART ASSOC. CLASS	NUMBER OF CASES	OPERATIVE DEATHS	LATE DEATHS	SURVIVORS				
				MARKEDLY IMPROVED	IMPROVED	SAME	WORSE	PERCENTAGE IMPROVED OR MARKEDLY IMPROVED (%)
I	2	0	0			2 (100%)		
II	12	1 (8.3%)	3 (25%)	1 (12.5%)	5 (62.5%)	0	2 (25%)	75
III	30	6 (20%)	5 (16.6%)	3 (15.8%)	14 (73.6%)	1 (5.3%)	1 (5.3%)	89.4
IV	3	0	0	1 (33.3%)	2 (66.6%)	-	-	100
Total	47	7 (14.7%)	8 (17%)	5 (15.6%)	21 (65.6%)	3 (9.4%)	3 (9.4%)	81.2

TABLE VII. CAUSES OF DEATH IN PATIENTS OPERATED UPON FOR PURE AORTIC STENOSIS BY THE TRANSAORTIC TECHNIQUE

Cardiac arrest (3 with severe hemorrhage)	4
Ventricular fibrillation	1
Uremia	1
Creation of severe insufficiency	1
Total	7

Even though 3 of these deaths are believed to be due largely to inexperience of the operator with this new technique, both the early and later operative mortality has been distinctly lower than that associated with the transventricular approach. Furthermore, a detectable increase in a preoperatively recognized element of aortic insufficiency was noted in only 1 patient in this series. Follow-up has been possible in all of those surviving surgery but only in 57 per cent has this follow-up been longer than six months.

The operative mortality experienced in 47 consecutive operations by the transaortic approach for aortic stenosis alone is expressed in Table VI and VII. That in 14 consecutive combined operations using this approach and a right-sided mitral technique is shown in Table VIII.

TABLE VIII. OPERATIVE MORTALITY IN TRANSAORTIC APPROACH AND RIGHT-SIDED MITRAL COMMISSUROTOMIES IN COMBINED MITRAL AND AORTIC STENOSIS

AMERICAN HEART ASSOCIATION CLASS	NUMBER OF CASES	HOSPITAL DEATHS	OPERATIVE MORTALITY RATE
I	0	0	0
II	5	0	0
III	9	1	11.1%
IV	0	0	0
Total	14	1	7.1%

Alterations achieved by surgery with respect to various objective findings and subjective symptoms are detailed in Tables IX and X.

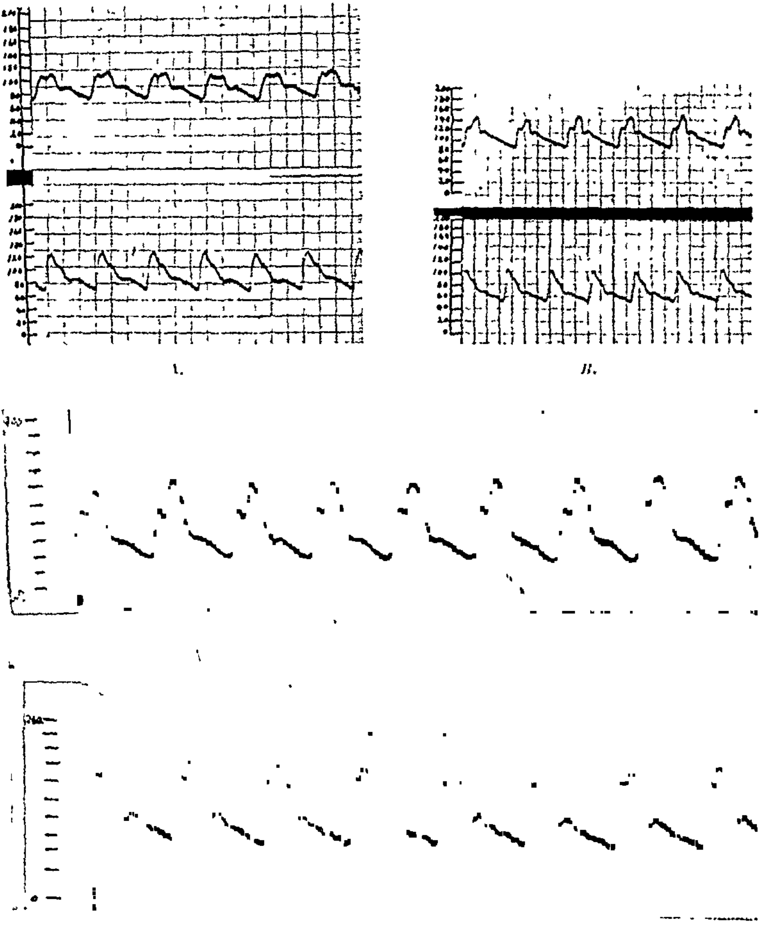
TABLE IX. SIX-MONTH FOLLOW-UP EVALUATION OF PRE- AND POSTOPERATIVE SYMPTOMS OF PATIENTS OPERATED UPON FOR "PURE" AORTIC STENOSIS (SUPRAVALVULAR APPROACH)

	DYSPNEA	SYNCOPE	FATIGUE	PAROXYSMAL NOCTURNAL DYSPNEA	ORTHOPNEA	ANGINA	PERIPHERAL EDEMA	VERTIGO
Present Preoperatively	90%	47%	81%	33%	28%	43%	24%	43%
Postoperatively	Improved or Absent 62%	Present 10.5%	Present 33%	Present 9.5%	Present 14%	Present 4.8%	Present 9.5%	Present 14%

TABLE X. OBJECTIVE POSTOPERATIVE CHANGES FOLLOWING AORTIC COMMISSUROTOMY (AT LEAST BY SIX MONTHS)

CHANGES POSTOPERATIVELY	AORTIC SYSTOLIC MURMUR (%)	AORTIC DIASTOLIC MURMUR (%)	AORTIC SECOND SOUND (%)	HEART SIZE X-RAY (%)	PULSE PRESSURE (%)
Absent	-	18.2	-	-	-
Diminished	54.5	4.5	-	13.6	5.3
Unchanged	41	45.5	31.8	72.8	52.6
Increased	4.5	31.8	68.2	13.6	42.1

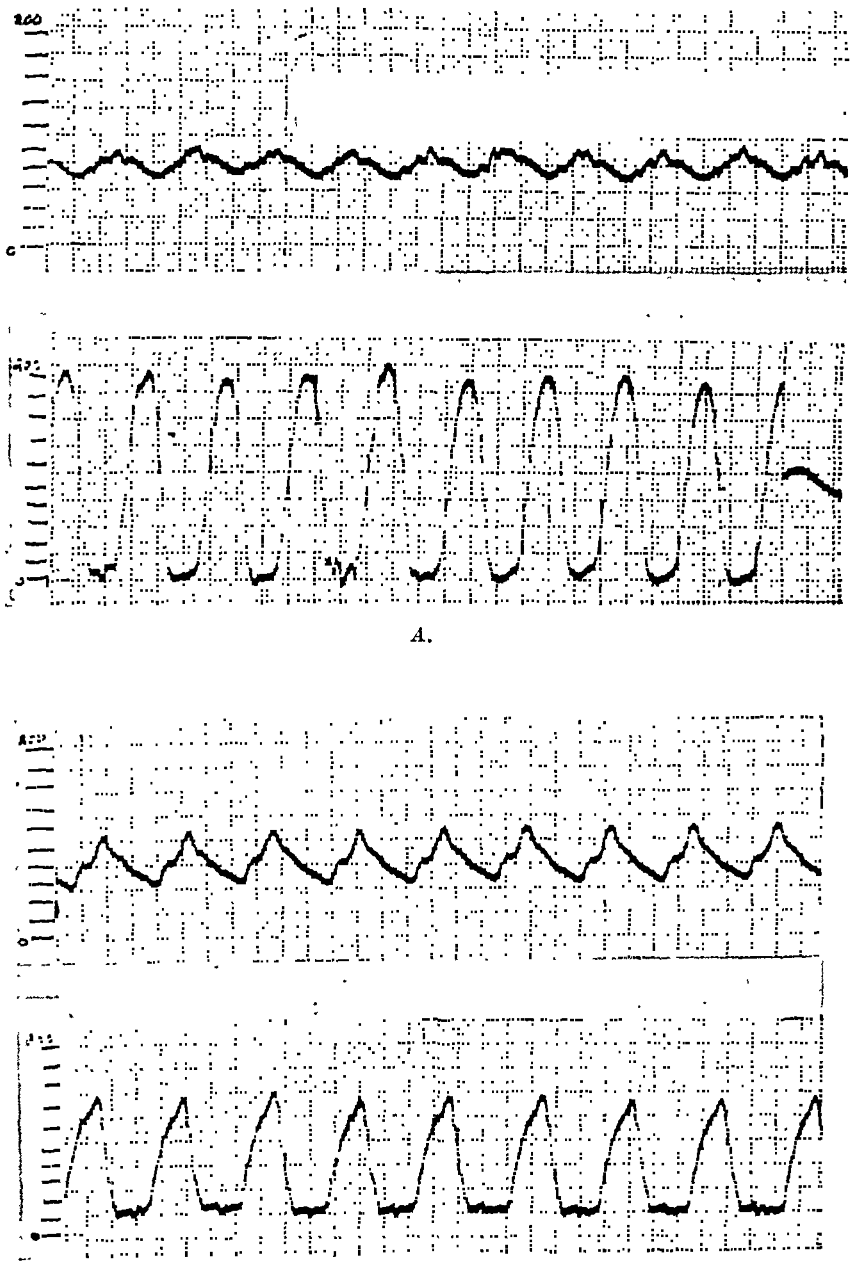
Alterations imparted to the brachial arterial pressure curves are shown in Fig. 27, *A*, *B*, *C*.



A. *B.*

C.

Fig. 27.—*A*, Abolition of the anacrotic notch (double-summit curve) in brachial arterial pressure tracing by satisfactory aortic commissurotomy. Upper curve, preoperative; lower curve, postoperative. *B*, Abolition of the anacrotic notch on the ascending limb of the brachial arterial tracing by satisfactory aortic commissurotomy. Upper curve, preoperative; lower curve, postoperative. *C*, Rise of the anacrotic notch on the ascending limb of brachial arterial tracing from a lower to a higher level, following aortic commissurotomy. This is considered as evidence of improvement (lessening) in the aortic stenosis although it is evident that a significant amount persists. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)



A.

B.

Fig. 28.—Precommissurotomy. *A*, Preoperative differential between aortic and ventricular pressures in W. E. (obtained by direct puncture at surgery).

B, Comparative postoperative pressures in W. E. showing abolition of gradient, obtained immediately after supravalvular digital aorbic commissurotomy.

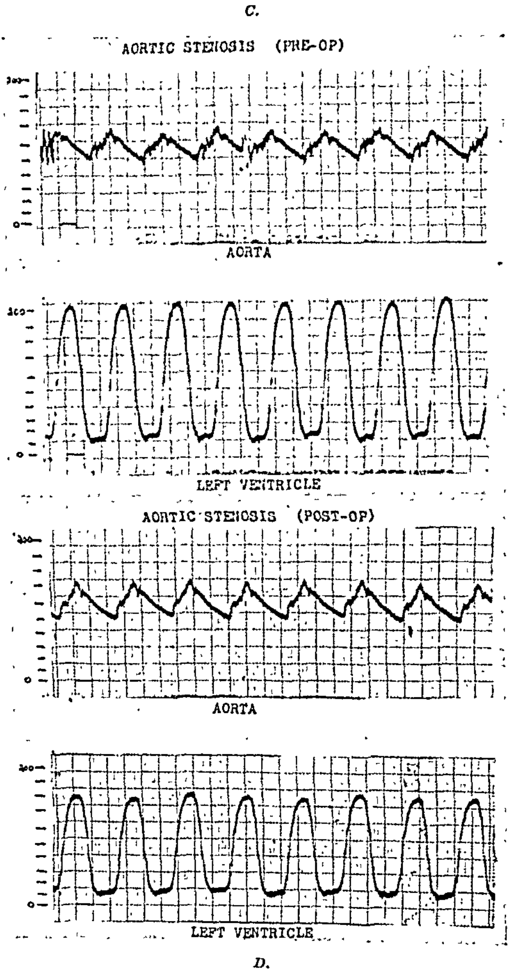


Fig. 28, *C* and *D*.—*C*, Pressure tracing obtained from the aorta and left ventricle in N. F., prior to aortic commissurotomy.

D, Comparative postoperative tracings showing reduction in systolic gradient but failure to achieve complete abolition by performance of supravalvular instrumental commissurotomy.

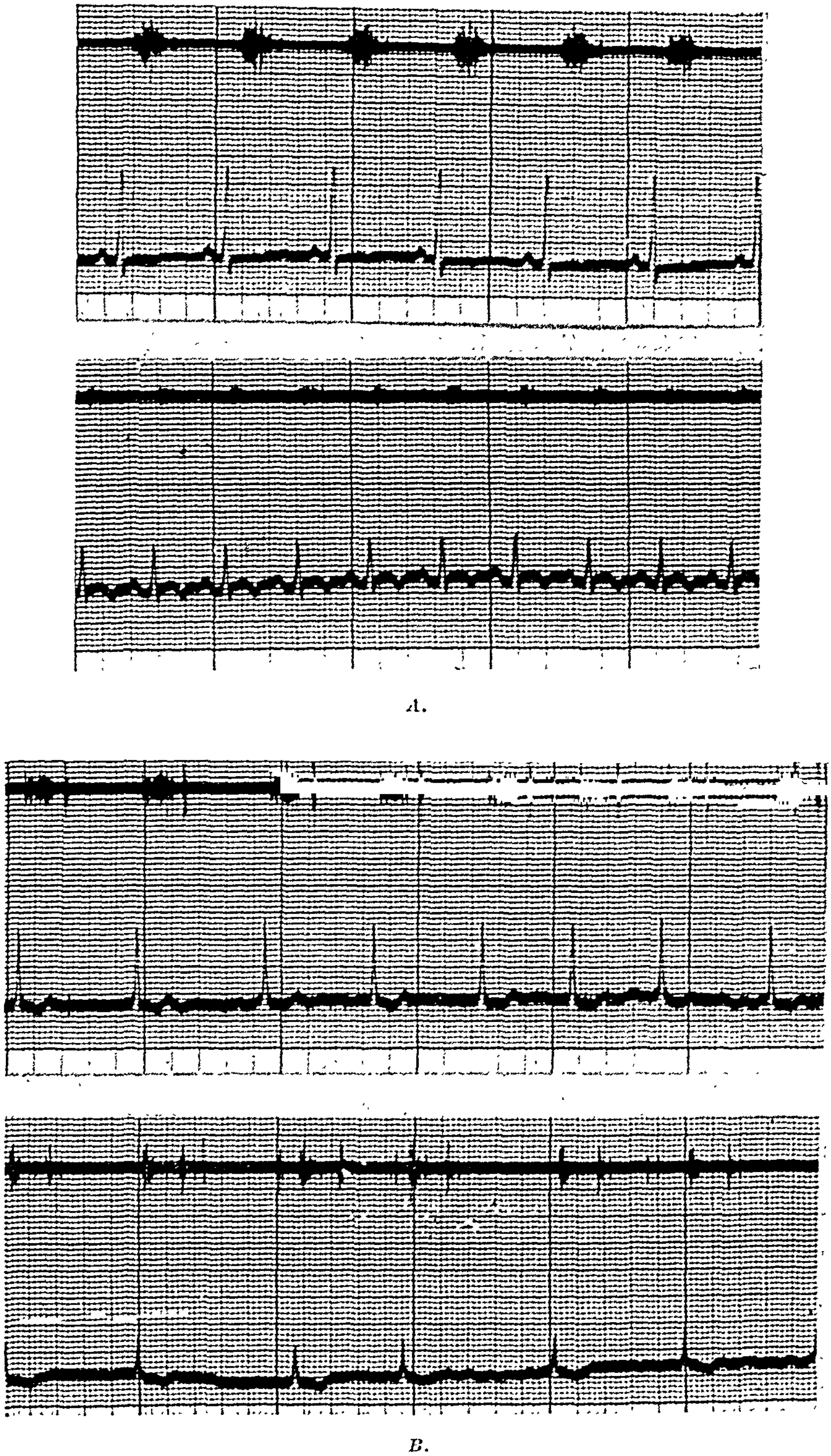


Fig. 29.—*A*, Phonocardiograms in A. W., showing a great postoperative reduction in the intensity of the systolic murmur of aortic stenosis. There is no evidence of restoration of the aortic second sound. (Upper part, preoperative; lower, postoperative.)

B, Phonocardiograms in L. M., showing a reduction in the intensity of the aortic systolic murmur and intensification of the aortic second sound following aortic commissurotomy. (Upper part, preoperative; lower, postoperative.)

Reduction in pressure differential between the left ventricle and the aorta (measured directly during surgery) are shown in Fig 28, *A-D* Unfortunately, we have not performed left heart catheterization in any postoperative patient as yet

Radiographic changes in heart size have been relatively minimal However, since most of these patients still maintain some residual element of aortic stenosis, even if it is only that degree which is related to stiffness of perhaps extensively mobilized cusps, it would seem unreasonable to expect great reduction in the size of the left ventricle

The phonocardiogram often shows a great reduction in the intensity of the basal systolic murmur and a return of the aortic second sound However, there is seldom a complete abolition of the murmur (Fig 29, *A*, *B*)

The spectrocardiograms[15] taken before and after aortic commissurotomy show a similar favorable alteration (Fig 30)

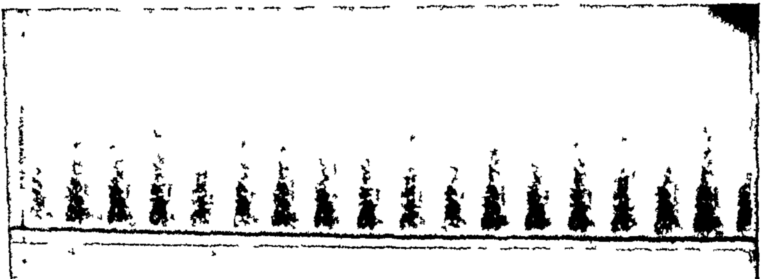


Fig 30 —Spectrocardiograms showing a reduction in the intensity of the systolic murmur of aortic stenosis and a restoration of the previously imperceptible aortic second sound following aortic commissurotomy in Patient F McN (These tracings were prepared by Dr George D Geckeler Chief of Cardiology at Hahnemann Medical College and Hospital Philadelphia Pa)

SUMMARY

1 A description of the pertinent pathologic and physiologic abnormalities observed in aortic stenosis has been presented

2 We have detailed the development of concepts and techniques of surgery for the alleviation of aortic stenosis

3 The indications for and steps in management of patients undergoing aortic commissurotomy are discussed

4. The improved techniques of management of isolated aortic stenosis and of combined mitral and aortic stenosis are illustrated.
5. The results of our experience to date are epitomized.
6. Trivalvular (mitral, aortic, tricuspid) exploration is recommended by way of a right-sided (third or fourth anterior intercostal space) incision whenever possible in all patients operated upon for a single rheumatic stenotic valve lesion. All correctible lesions found should be treated at the same operative session.

CONCLUSIONS

Severe rheumatic aortic stenosis is a medically intractable and often a promptly fatal illness. However, some patients may survive for years, although with increasing incapacitation. This lesion is reasonably responsive to appropriate surgical separation of the obliterated commissures.

This should be performed, whenever feasible, by the transaortic route, using digital "vision" to explore the valve and to control the course of any instrumentation. Splitting of one or more fused commissures by simple finger pressure is possible in almost one-half of the patients. Instrumental aid is mandatory in the rest.

Coexisting mitral and tricuspid stenosis may be approached and relieved satisfactorily from the right side through the same right anterior thoracic incision. Indeed, it is now our custom to perform elective surgery for "isolated" mitral stenosis by the right-sided thoracic approach largely in order that any other valvular conditions unexpectedly encountered may be treated simultaneously.

The operative mortality and the clinical improvement obtained in surgery for aortic stenosis now closely approach the satisfactory levels established in commissurotomy for mitral stenosis.

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DISCUSSION

DR. HARRISON BLACK, Boston, Mass.—I should like to congratulate Dr. Bailey on his large series but would like to go back to the discussion on aortic stenosis where we started. Our interest on Dr. Harken's services in Boston has been directed, during the developmental phases of aortic surgery, toward determining which patients need surgical relief. I believe that as experience increases one comes to realize that one swallow does not make a summer and that a murmur and thrill (caused by turbulence of blood) do not necessarily indicate a hemodynamically significant valvular lesion. Therefore, up to the present we have chosen to operate only when aortic stenosis has produced part or all of the triad—angina, syncope, or left ventricular failure. When mitral and aortic stenosis have coexisted, we have not corrected the aortic lesion unless the transaortic gradient measured on the table exceeded 50 mm. Hg. (slide) Our choice of this figure is arbitrary but is based on the pressure-work studies of Dr. Richard Gorlin of our Medical Department. (Bull. of New England Med. Center 16: 13-23, 1954.)

On the diagram you will note that the work of the left ventricle rises steeply when the gradient exceeds 50 mm. Hg. In a group of between 30 and 40 patients with such combined lesions but with an aortic gradient below 50 we have performed mitral valvuloplasty only. The follow-up of these patients over several years apparently justifies this decision, for the vast majority have been markedly improved and have been able to return to a normal level of activity. From this we may conclude that some patients with mitral stenosis and an aortic murmur and thrill may do as well without the aortic operation as with it.

(slide) Dr. Gorlin's study has documented another important point that has become increasingly apparent from clinical experience, that is, that the correction of aortic stenosis cannot be accomplished by the creation of insufficiency any more than is true in dealing with the mitral valve. This diagram reveals that with an aortic valve of a given area the work of the left ventricle increases threefold when insufficiency is added to the stenosis.

Our record with the transventricular approach to the pure aortic stenosis is quite similar to Dr. Bailey's with approximately 30 per cent early mortality and less than 50 per cent survival when the late deaths are included. For this reason we have turned to the transaortic approach described by William Swann of Knoxville. This is vastly better tolerated than the transventricular operation since neither pleural cavity need be opened, both lungs are expanded and available for ventilation throughout the procedure and the problems of ventricular cardiotomy (hemorrhage, standstill, and ventricular fibrillation) are avoided. We use a sternal splitting approach and I think Dr. Bailey will be intrigued by the opportunity this offers to operate on four valves at one sitting.

(slide) In our experience the vast majority of stenotic aortic valves are calcified by the time they become symptomatic and are bicuspid due to fusion of the two anterior cusps. Rarely have we seen the type so commonly pictured in pathology texts—those with an equal fusion of all three commissures. At the time of our first transaortic operation the importance of avoiding a fracture through the heavily calcified anterior commissures was not adequately appreciated and the patient died on the table from embolization of calcified particles. In 12 subsequent cases, fracture with the finger or breadknife valvulotome has been limited to the lateral commissures and extended along the aortic wall anteriorly and posteriorly on each side. By this means a double "trap-door" valve is created without risking embolization and without the production of insufficiency. The calcific bars are not broken into but mobilized as a unit.

The only additional death from a transaortic operation occurred in a woman with advanced coronary artery disease in addition to aortic stenosis. In this case, as in several others, the transaortic gradient has been entirely eliminated. As experience is gained with this procedure and its safety established, we will probably accept for operation individuals in whom the gradient is above 30 or even 20 mm. Hg. We are going to have to accept patients earlier for aortic stenosis surgery in order to correct the lesion before calcification adds to the risk and seriously interferes with the restoration of competent valve function. To do so undoubtedly will depend on a reliable method of measuring the transaortic gradient before operation.

DR. CONRAD LAM, Detroit, Mich.—I should like to show the post-mortem specimens of the aortic valves of 3 patients which show what may happen when a dilator of the Bailey type is inserted and opened.

(slide) This illustrates a technically successful aortic commissurotomy. The patient was near death from aortic stenosis at the time of the operation and, unfortunately, had adhesive pericarditis which made some dissection necessary before the aortic root could be palpated. The heart was in standstill when the transventricular operation was begun. After the dilatation, the heart was easily resuscitated, and the patient lived for four days with a normal blood pressure. Death resulted from the residuals of cerebral anoxia.

(slide) Following dilatation of the aortic valve in this patient, a piece of tissue was found caught in the blades of the dilator. It was feared that this represented one of the cusps, and as the failing heart was massaged, a diastolic thrill indicative of marked aortic insufficiency could be felt. The post-mortem specimen showed that a piece of the valve had been removed.

(slide) This valve was dilated, but because it was of the congenital type, it did not tear or open up in a permanent fashion. Death resulted from a delayed rupture of the left ventricle, apparently at the site of a laceration made by the guide wire as it was inadvertently forced against the opposite ventricular wall. A valvotomy of the Brock type would have been indicated for this valve. Probably it could have been accomplished with a cutting instrument such as the Bailey mitral guillotine.

DR. WILLIAM SWANN, *Knoxville, Tenn.*—We would like to show a slide which is now almost two years old. It was previously shown before this Association one year ago at Montreal in the course of a paper "Intracardiac Surgery with the Aid of Artificial Operative Tunnels." When we showed this slide, we described both finger fracture and instrumental incision of the aortic commissures. We believe this was the first time that this operative technique in relation to the aortic valve had been described.

The first patient was operated upon in June, 1953, and was a patient upon whom Dr. Gerbode had previously operated for mitral stenosis. When we operated on the first patient, we knew that Dr. Bailey had, two months earlier, already used the dilator method from above, through a pericardial pouch.

The actual opening through the fused aortic commissures either with the finger or the knife would seem to be the preferable method. This has been graphically illustrated by McMillan of London with motion pictures of the valve before and after the use of the dilator, and before and after accurate incision with the knife.

(slide) This slide illustrates our present preference, the guillotine knife, and also shows the single channel operative tunnel presently used by us. It is fashioned in two layers. There is an inner layer of plastic for ease of maneuver and an outer layer of cotton cloth to lend strength and particularly to aid in hemostasis at the suture line. As to the substance that is used for the auricular appendage substitute, that is a matter of personal preference. We have, at the invitation of Dr. Bailey, been privileged to observe his use of the pericardial pouch. In his hands this method is satisfactory. In our hands the method is unnecessarily time consuming, difficult, and not entirely dependable. Certainly there is no advantage to fashioning the operative tunnel, or pouch, from living tissue inasmuch as it is to be discarded at the end of the procedure. A pouch made of rubber or plastic alone is not satisfactory. Dangerous leakage occurs at the suture line. A fabric such as cotton cloth should be incorporated, at least at the line of suture to the aorta.

DR. I. M. MADOFF, *Boston, Mass.*—I am certain that the technique which Dr. Swann has described and Dr. Bailey has so successfully carried forward will eventually prove to be an extremely useful procedure. However, with the thought that some versatility in the technical approach to the treatment of aortic stenosis might produce better over-all results, I would like to review briefly an approach in which we have been interested. This approach is derived from a study of freshly autopsied heart specimens and experiments on anesthetized dogs. As you know, the aortic valve lies in relationship to the left atrium and the right atrium posteriorly and laterally. By inserting the finger into the left atrium, as one does in operating for mitral stenosis, and invaginating the wall of the right atrium at the same time, one can manipulate and compress the aortic valve ring and indirectly change the contour of the valve leaflets. In some instances of aortic valvular stenosis, we have noted that, despite adequate dilatation of the stenotic valve by various instruments, the valve lumen remains the same even though the commissures appear to be separated. However, by compressing the valve ring externally in this manner, the valve ring becomes more mobile and the size of the valve lumen increases. This maneuver will produce an increase in blood flow through the aortic valve area, not only by means of increasing mobility of the valve ring, but also by increasing the size of the valve lumen.

(slide) This shows a typical aortic valvular stenosis with calcification.

(slide) The finger is introduced into the left atrium, the right atrial appendage is inverted and you notice that the valve ring is compressed.

(slide) At the completion of the maneuver this shows the dilated valve orifice to be much larger.

(slide) Since one may potentially further narrow the valve lumen temporarily by compression of the valve ring this instrument, called a flowtome, has been designed. This permits blood flow through the aortic valve at least sufficient for coronary circulation while the compression maneuver is being accomplished. The instrument is introduced through the left atrium and its head is brought out through the aortic valve.

(slide) This is the flowtome in situ prior to compression.

(slide) This shows the compression maneuver with the flowtome in position and how it can permit continuous blood flow.

(slide) At the completion of compression you will notice the significantly dilated aortic valve lumen.

Some fifty heart specimens with aortic stenosis have been examined; in these, this procedure has produced effective aortic valve dilatation in 3 out of 4 cases. However, it can be accomplished without many of the recognized hazards of the other surgical approaches to this lesion. If the procedure is found to be not effective as determined by pressure gradients observed at operation, in a given case, one may proceed with one of the other available methods.

DR. CHARLES P. BAILEY (Closing).—I want to thank the discussants and will try to answer their questions. I well remember previous discussions with Dr. Harken on the subject of aortic stenosis and possible suitable surgery. I am sorry he and Dr. Black did not choose to follow my lead until Dr. Swann followed it and reported corroboration.

I know that abnormal auscultatory findings over the aorta are not necessarily indicative of physiologic abnormality but, on the other hand, they may be. I would like to show the first slide to clarify what I meant when I said there might be a difference between the differential in systolic pressures obtained between the aorta and the left ventricle, preoperatively, and those in the same case obtained directly during operation. You see the marked reduction in the differential pressures, probably because of diminution in the cardiac stroke output, due perhaps to anesthesia, the open chest, and in part to blood loss. Also, after the obstruction of a mitral valve is opened in a case of combined aortic and mitral stenosis there might be difficulty in showing a significant gradient physiologically since the curves may be "damped" due to anesthesia or to the depression of surgery. By left and right combined heart catheterization usually one can tell if the aortic stenosis is significant preoperatively. I think it has become fairly evident by now that if one does not relieve coexisting aortic stenosis when a stenotic mitral valve is opened, he does the patient great disservice, because there is then obstruction of the outflow valve of the left ventricle but no longer of the inflow. The patient therefore has essentially isolated aortic stenosis, which is a much more malignant disease than is the combination of the two stenotic lesions I think, however, it would be good to have evidence of a pressure differential of at least 30 mm. before opening the aortic wall to palpate the aortic valve through some kind of pouch.

We do not agree that all aortic valves have become bivalvular when they are calcific We know that less than one half of them are bivalvular when they are calcific, and we think that at least 90 per cent of aortic valves operated upon for stenosis are calcific at the time of surgery.

Dr. Lam showed rather beautifully in a few cases how transventricular aortic dilatation can help the valve, and in one how a technical complication can produce insufficiency and cause death of the patient.

(slide) This slide indicates why we gave up the transventricular route to the aortic valve and, of course, the inadvertent creation of regurgitation was one of the reasons. This is a blind, purely instrumental, operation. Since apparently a finger is necessary for the accurate splitting of the commissures, the supravalvular approach certainly is preferable.

Dr. Swann evidently used the guillotine knife before we did, although I am sure we had split the valve digitally as well as dilated it with an instrument prior to that time The suggestion that a pouch might be used to enter the aortic lumen had been made by Dr. William Glenn prior to the time we used it. He actually used it in experimental animals

(slide) This slide shows the cause of death in 19 patients operated on by the transventricular route. I think you can see that most of these deaths are related to going through the ventricle which may be very soft, so that there may be uncontrollable hemorrhage, and very irritable so that there may be ventricular fibrillation or cardiac arrest. Either of these rhythmic changes sometimes may be related to the inadvertent creation of aortic insufficiency.

Dr. Madoff's operation is very interesting and, of course, might prove in some cases to be a very great help.

MANAGEMENT OF THE POSTRESECTION SPACE IN TUBERCULOSIS

II. Following Lobectomy

John W. Bell, M.D.
Sunmount, N. Y.

THE propensity with which tuberculosis affects the lung apex, whether postural, biochemical, or inherent, has directed the efforts of resection surgery primarily toward the upper lobes. Thus, upper lobectomy has become a relatively common operation in the repertory of the thoracic surgeon. In patients without evidence of endobronchial disease or resistant tubercle bacilli, lobectomy was proved to be a safe and often curative procedure. Current reports.[1-6] including our own experience with segmental and subsegmental excision, indicate a definitely higher nontuberculous morbidity than is found with lobectomy.

We shall not discuss the indications for lobectomy, nor the relative function-sparing advantage of saving the right anterior segment or lingula on the left. Rather, we are concerned with the problem of a neutral or positive pressure postlobectomy space and the success of right middle and lower lobe remnants in filling the hemithorax.

The elimination of residual intrathoracic space is not entirely accomplished by expansion of adjacent parenchyma. Relative shifting of the superior mediastinum (Fig. 1), narrowing of the upper interspaces, and a temporary rise in the hemidiaphragm, all serve to reduce the volume requirement of the upper chest. Moreover, the resumption of space relationships by middle and lower lobes is not always an acute process. Upper lobes, harboring enough tuberculosis to require their complete excision, vary widely in size. At operation, one may encounter a range from normal volume to a tiny shrunken paramediastinal wedge occupying one-eighth or less of the hemithorax (Fig. 2). As the upper lobe progressively contracts, even with chest wall and mediastinal compensation, some expansion of middle and lower lobe tissue takes place. Commonly, the right middle lobe or lingula, and the superior segment of the lower lobe, are the units contributing most to this volume readjustment.

Turning from the mechanical considerations, the physiological adjustment, subsequent to lobectomy, may also be minimal. This, in part, is due to the fact that essentially functionless tissue is removed. In the diseased area, although ventilation is reduced less than circulation and oxygen uptake, the net change reflected in pulmonary function studies after full recovery may differ little from preoperative values.[7-9]

From the Veterans Administration Hospital, Sunmount, N. Y.

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MATERIAL

During the five-year period, Jan. 1, 1950, to Dec. 31, 1954, 135 lobectomies were performed at the Veterans Administration Hospital, Sunmount, N. Y. (Table I). A review of this material discloses 30 cases (22 per cent) of postlobectomy space requiring clinical recognition, and, in most instances, supplementary treatment. We have included in this group cases in which superior segmental resection or wedges from middle or lower lobes was accomplished concurrently with the upper lobectomy. Superior division resection on the left, although the counterpart of a right upper lobectomy, was not included in this series.

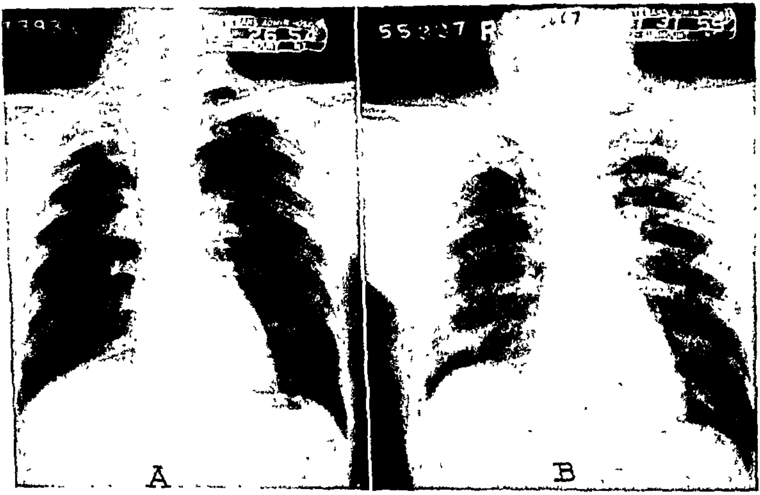

Fig. 1.—*A*, Preoperation. *B*, 8 months postoperative, right upper lobe.

Table I. Postresection Spaces Following Lobectomy—135 Operations January, 1950, to December, 1954

NUMBER OF PATIENTS	CAVITY STATUS PREOPERATIVELY		SPUTUM BACTERIOLOGY PREOPERATIVELY		LOCATION OF SPACE		DATE OF SPACE APPEARANCE POSTOPERATIVELY	
30	Closed	10	Negative	21	Apical pneumothorax	18	1st week	26
					Apical hemothorax	2	2nd week	2
	Open	20	Positive	9	Basal pneumothorax	5	3rd week	1
					Combined (apical and basal pneumothorax)	3	4th week	1
					Apical intrapleural space communicating with subscapular	2		

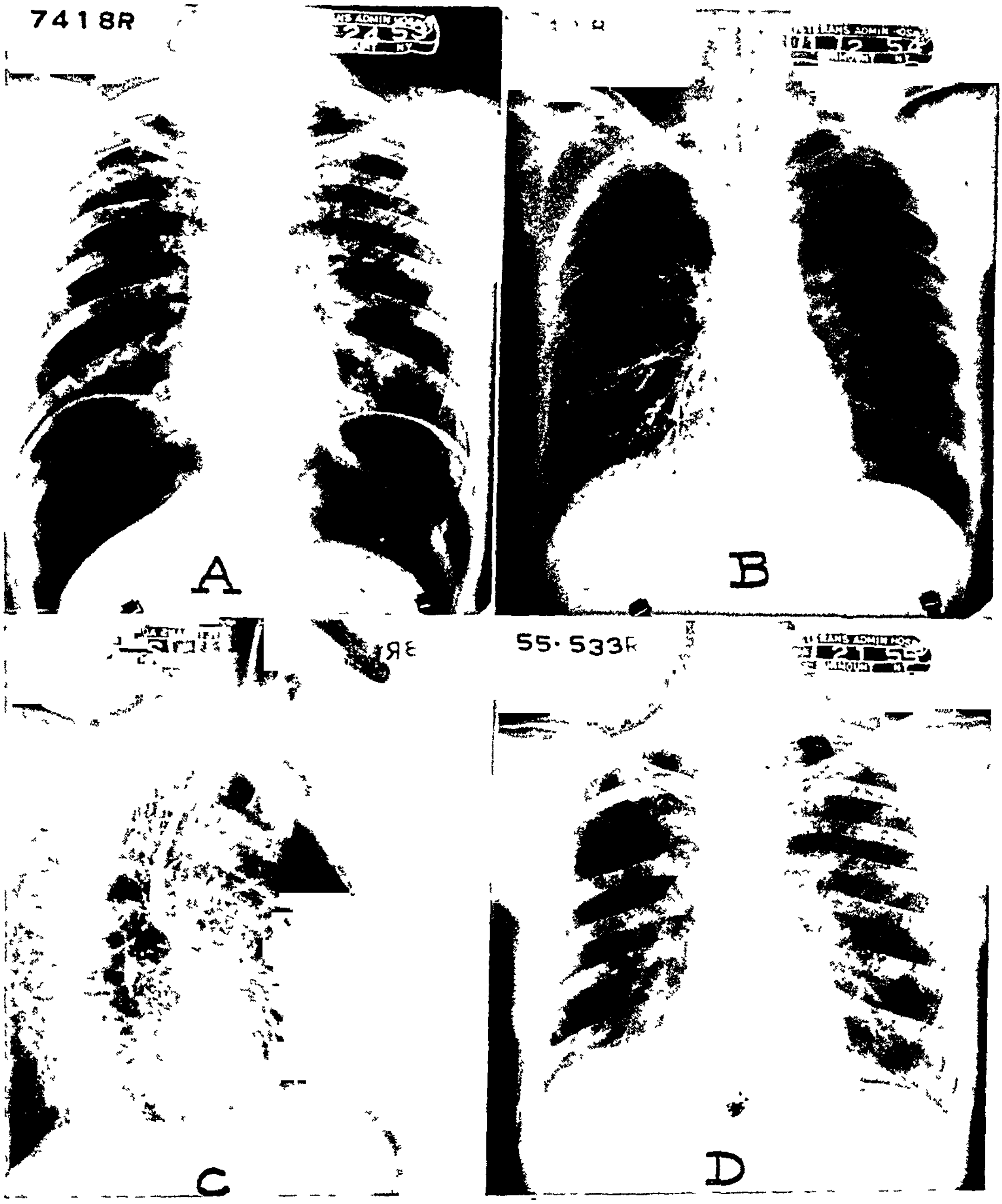

Fig. 2.—*A*, Preoperation. *B* and *C*, Bronchograms. *D*, Postoperative, right upper lobe.

The addition of segmental or subsegmental excision to lobectomy introduces the factors of complications incident to this type of operation. The occurrence of postoperative air leaks may parallel the number of segmental stumps and absolute amount of raw lung surface remaining after such parenchymal barnstorming.

Segments or subsegments from the lower lobe also influence the location of the postresection space (Table I). Air leaks from these areas are prone to result in lateral or supradiaphragmatic pneumothoraces. All basal spaces in our group occurred in cases where segmental or multiple subsegmental excision was added to the upper lobectomy.

The lobes removed in this series represent a variety of moderately and far-advanced tuberculosis, with a cavitary area existing or suspected in the majority. The sputum was almost equally divided between the positive and negative states. As with segmental resections, the preoperative disease state was not found to be related to the frequency of postresection nontuberculous complications.

OPERATIVE MANAGEMENT

Anticipation of a space problem begins at the operating table. An upper lobe of subnormal volume simplifies the problem, as compensation has already begun in adjacent lobes. The physical appearance of the remaining lung is most important. Slow inflation, trapping, or delayed emptying, or any aberrations in ventilation, may foretell difficulties in postoperative expansion. The extent of distribution of residual disease is significant. Fine seeding throughout the parenchyma, stellate areas of fibrosis, with surrounding patchy areas of obstructive emphysema, complicate re-expansion.

Initially, we felt great trepidation in expecting prompt re-expansion of lower lobes containing moderate amounts of residual disease. These were cases in which so-called compromise resection surgery was carried out. The cavity-bearing upper lobe, with adjacent nodular disease, might represent 60 to 70 per cent of the total disease in that lung. The presence of contralateral disease, and other considerations, made retention of the lower lobe or basilar segments almost a necessity if resection was at all feasible. We were most pleased to observe prompt expansion and occupation of almost the entire hemithorax by lower lobes or basilar segments alone which contained various amounts of nodular disease (Fig. 3).

Following the surgeon's estimate of predictable space filling, several measures are available to aid this readjustment. These measures may be divided into two groups; those accomplished within the chest and those applied to the chest wall. Several thoracic surgeons, following the suggestions of Blades, as an adjunct in the Blalock operation, divide the inferior pulmonary ligament to allow ascent of the hilum. This appears to be a useful addition, particularly after left upper lobectomy, but it is doubtful that it is routinely necessary. Any effort to paralyze the hemidiaphragm temporarily is mentioned only to be deplored. One of the main problems in the postoperative period is the proper raising of secretions. Paradox of this muscle not only interferes with ventilation, but robs the dependent lower lobe of the pistonlike action which is vital for an effective cough mechanism. Far better, from the physiologic standpoint, is the institution of a pneumoperitoneum postoperatively, when it is desired to raise the level of the diaphragm.

The creation of a so-called apical pleural tent, as described by Miscall,[10] is a valuable adjunct. The fashioning of a satisfactory tent, of course, depends upon the integrity of the parietal pleura. Any entry into the extrapleural plane, during mobilization of the upper lobe, may make such a tent impossible. In addition, a rent in the extrapleural or subcostal tissues mitigates against concomitant freeing of the subcostal or extraperiosteal tissues, or against any variation of thoracoplasty. Such openings, even when sutured, may open during the stress of local paradox, allowing the intrapleural and subscapular spaces free communication. In the event of a subsequent bronchopleural fistula, with pleural infection, this sepsis, either pyogenic or tuberculous, may involve the subscapular tissues.

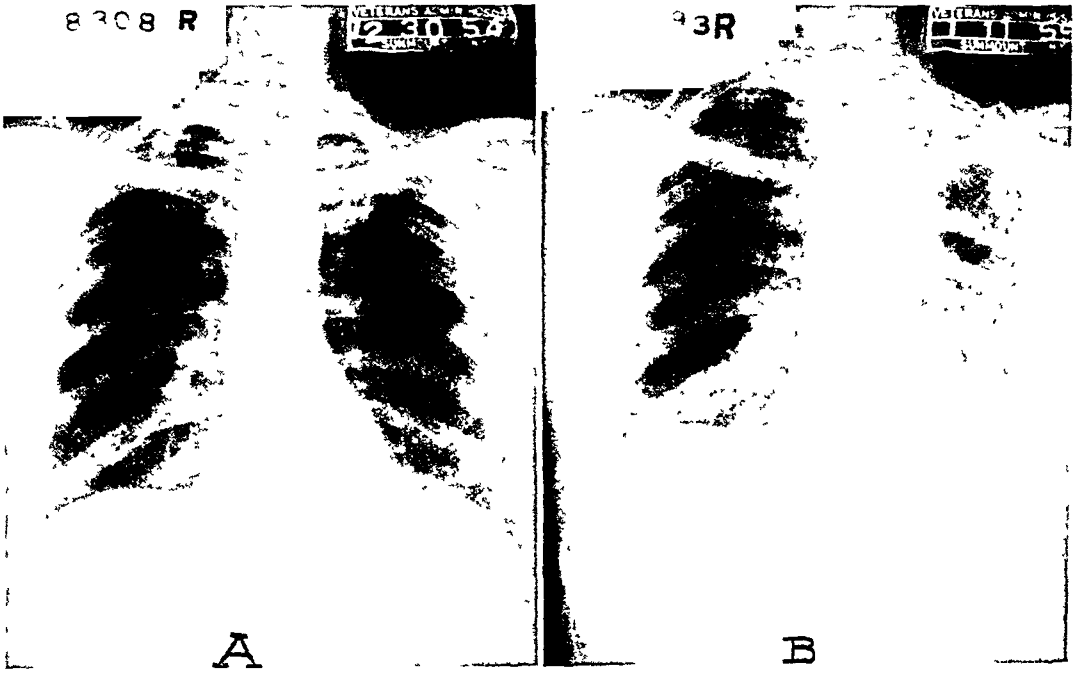

Fig. 3.—*A*, Preoperation. *B*, 6 days postoperative, left upper lobe and superior segment, left lower lobe.

An apical pleural tent may aid in pleuralizing and thus sealing raw lung surfaces of the middle or lower lobes. This same advantage may be secured by mobilization of the subcostal tissues as used by Chamberlain and associates,[1] without removing or altering the adjacent ribs. In this situation, some surgeons have introduced a small plombage into the subcostal space. Our experience with 4 cases of concomitant plombage have been uniformly poor. Each patient became infected, and required subsequent removal and completion of thoracoplasty. In view of the 25 per cent incidence of tuberculous infection, reported by Gibbons, Desforges, and Strieder,[11] in formal plombage with presumably intact extraperiosteal tissues, we believe the insertion of a concomitant foreign body at the time of resection is unwise.[12]

Various modifications of conventional thoracoplasty are in current use. Steele and co-workers[4] have described the division of the upper ribs into short segments without removal as an aid to reduce paradox. Bjork's[13] description of a concomitant osteoplastic thoracoplasty may be a most valuable procedure when a simultaneous maneuver is necessary to reduce intrathoracic space.

Our modifications and indications for the pre- and postresection thoracoplasty and the pleural tent, each comprising a group of approximately 25 cases, will be discussed in the next and last study on the management of the postresection space in tuberculosis.

TREATMENT

Assuming that no type of concomitant procedure was carried out with the resection, the treatment of a postoperative pneumothorax is contingent upon the degree of air leak, if any, contributing to the space.

The majority of space problems may be divided into two groups. In the first are apical and posterolateral caps, which on single or multiple aspirations demonstrate a neutral or slightly negative pressure relationship. Patients in this category usually have no overt symptoms of a fistula, i.e., absence of irritative cough, streaking, or fever. Moreover, the surgeon may, as a result of palpation or the behavior of the lung during operation, have reason to anticipate delay in re-expansion of the parenchyma. Retention of secretions or excessive air leaks in a moderately emphysematous lung, during the postoperative period, contribute to failure of space occupation. If unassociated with a hemothorax, such a space may not require any definitive treatment. Serial films may disclose an ever decreasing air cap. One may expect gradual expansion of the remaining lung and the eventual compensation of superior mediastinum, upper interspaces, and diaphragm, to reduce the hemithorax and eliminate any residual pneumothorax (Fig. 4). Uncertainty as to the presence or absence of a bronchial communication, if not resolved with manometry, may frequently be clarified by bronchography.[14]

Apical hematomas of any magnitude (two or more interspaces) are best evacuated by *secondary thoracotomy*. At this time, a concomitant chest wall procedure may be added to reduce the actual or potentially infected space. Other than the small, so called rim hemothorax, which may undergo spontaneous resolution, we have not been successful even with the assiduous use of enzymes in evacuating such collections of blood through a needle. Occasionally, with the use of an intercostal catheter, clearing of the space has been accomplished. However, an appreciable collection of clotted blood at the apex, with possible *intermittent fistulization*, is ripe for secondary or primary tuberculous infection. Such an apical empyema may be anticipated in some cases and surgically prevented.

The presence of a positive tension space in the postoperative period requires immediate treatment. Usually such a pneumothorax is manifest shortly after removal of the *primary thoracotomy tubes*. An x-ray at this time may show a so called blowing up of the upper hemithorax with concave depression of the lower lung and shifting of the superior mediastinum to the contralateral side

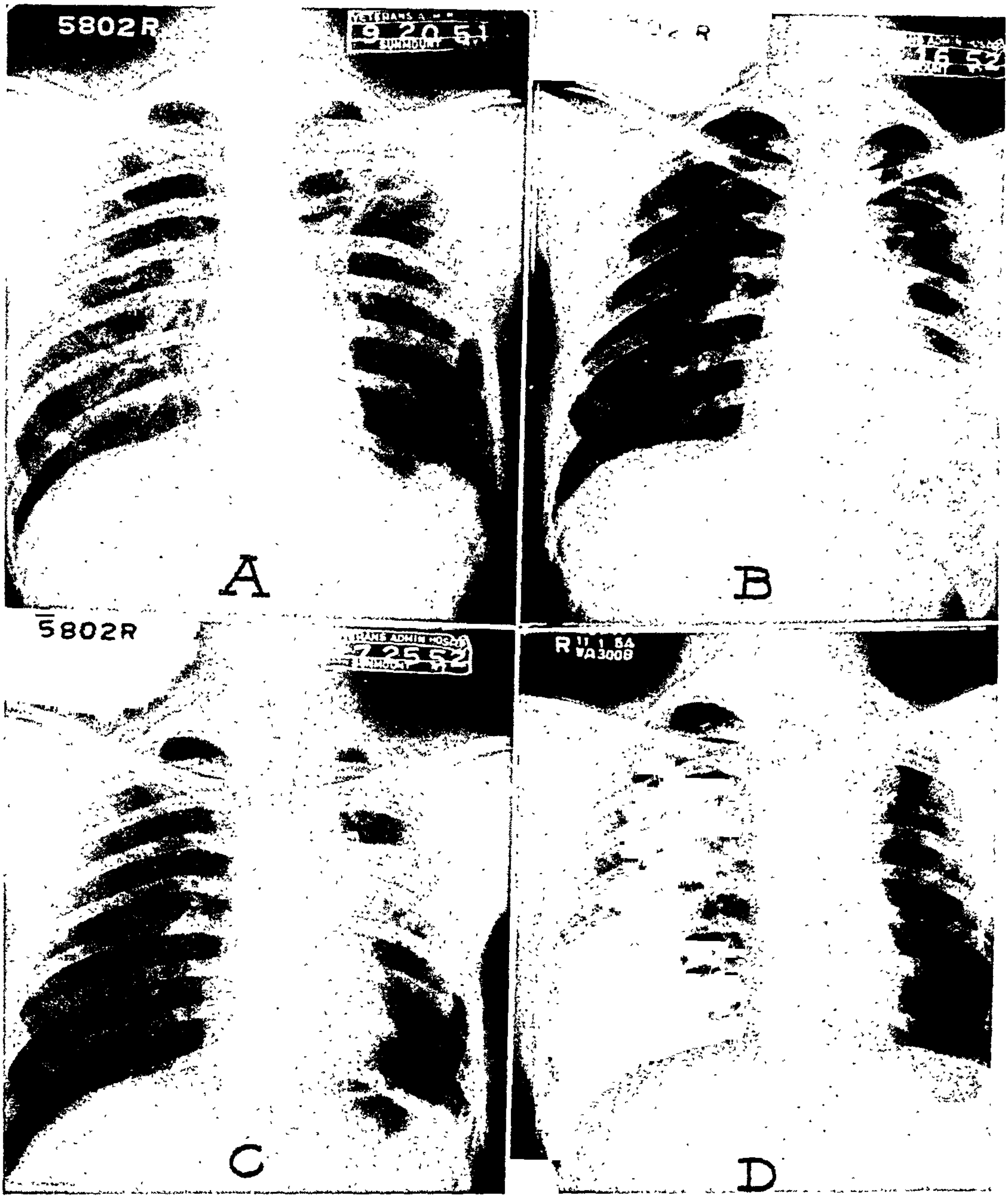

Fig. 4.—*A*, Preoperation. *B*, Postoperative, left upper lobe, showing left apical space. *C*, Persistent left pneumothorax 3 months postoperative. No treatment, no fistula demonstrated. *D*, Two and one-half years postoperative. Marked compensatory shifting of superior mediastinum.

The surgeon may now elect the following alternatives of treatment. Prompt intubation of the space will relieve any symptoms associated with the local tension pneumothorax. Closure of an underlying fistula of any magnitude usually will not occur, however, with tube drainage alone. The necessity of a space obliterating procedure for the inevitable empyema, in addition to the possibility of closing the fistula, may make the postresection thoracoplasty a logical step. Since accurate intubation of a subthoracoplasty space is difficult, prior insertion of a tube is of value. The tube may be maintained postoperatively until there is evidence of fistula closure.

Observation has shown that the presence of an intercostal catheter after approximately one week, even with constant antibiotic coverage, is nearly always associated with secondary infection. The occurrence of a postlobectomy fistula may then present the surgeon with the contrary treatment alternative, that of secondary thoracotomy and attempted revision of the bronchial stump. Since this procedure is much safer when risk of infection is minimal, prior intubation may be omitted.

If direct surgical closure of the fistula is successful, adjacent parenchyma may re-expand and cure is accomplished. Yet even in the absence of a positive space, the remaining lung may be incapable of occupying this area. Anticipation of this problem may lead the surgeon to perform a concomitant thoracoplasty at the time of secondary thoracotomy. Otherwise, chest wall surgery may be required as a third operation.

RESULTS

Review of treatment employed in this group (Table II) discloses that one-third of the patients were managed by intrapleural catheter drainage. These cases represented small transient bronchopleural fistulas, which closed after one to two weeks of underwater suction. The apical spaces in 2 patients required prolonged drainage up to five weeks, and were in each instance complicated by penicillin-resistant hemolytic *Staphylococcus aureus* infection. The fistulas closed satisfactorily; however, since tubercle bacilli were not recovered from the space, thoracoplasty was not added.

Pneumoperitoneum, with or without intubation of the chest, was used in 4 instances. This combination is useful, chiefly for the supradiaphragmatic space so prone to occur when superior segmental excision or multiple wedging of the lower lobe is added to the upper lobectomy.

Our employment of a concomitant thoracoplasty was limited to 2 cases. The first was actually a modified plombage, with the ribs retained and Gelfoam placed in the subcostal space. The subsequent development of an upper lobe stump fistula, which communicated with and infected the subcostal and subscapular spaces, led to removal of the ribs and appropriate drainage. The second case had no complications.

Our indications for postresection thoracoplasty in this group were the persistence of a large apical space, with or without an associated fistula, or the demonstration of a positive culture for tubercle bacilli from this space. The thoracoplasty as used in this manner was satisfactory in aiding fistula closure, although results were generally better when the thoracoplasty was done over an intrapleural tube which was gradually withdrawn as the fistula healed.

The results of space treatment are noted as cured or satisfactory in 21 patients. The unsatisfactory cases were largely due to the injudicious and prolonged use of catheter drainage alone, when the addition of a small apical thoracoplasty might have hastened or improved the end results.

The current status of tuberculosis in each patient with a postoperative pneumothorax is summarized (Table II).

TABLE II. TREATMENT OF SPACE PROBLEMS FOLLOWING LOBECTOMY (30 CASES)

TREATMENT		DURATION OF INTERCOSTAL DRAINAGE		INFECTION (PREDOMINANT ORGANISM)		RESULTS OF SPACE TREATMENT		STATUS OF TUBERCLE BACILLI (N.T.A.)	
No treatment	7	1 week	7	Hemolytic *Staphylococcus aureus*	2	Cured	12	Inactive	23
Thoracentesis	3	2 weeks	3					Active	3
Intercostal catheter	10	4 weeks	1	Tubercle bacilli	2	Satisfactory	9	Negative (In hospital)	1
		5 weeks	1						
Intercostal catheter with pneumoperitoneum	2	6 weeks	1	Mixed (staphylococcus and tubercle bacilli)	1	Unsatisfactory	9	Deceased (1 tuberculous; 1 nontuberculous)	2
Pneumoperitoneum	2							No follow-up	1
Concomitant thoracoplasty	2								
Postresection thoracoplasty	4								

COMMENT

A local postresection tension pneumothorax requires prompt clinical recognition. This situation, with the implication of a frank bronchial stump fistula, may be a surgical emergency equivalent to a nonsurgical spontaneous tension pneumothorax. Even very small or intermittent fistulization in the tuberculous patient may lead to an eventual pyogenic or tuberculous empyema in a persistent intrathoracic space.

Surgical decompression may follow two electives: multiple needle, or, if positive pressure is apparent, closed thoracotomy intubation of the space. This expedient will eliminate the symptoms of tension. Although open thoracotomy is theoretically always possible even after intubation, the chances of success in fistula closure and local débridement of the space diminish greatly as intubation is prolonged.

Following the diagnosis of a postresection space, if the surgeon has had good experience with fistula revision, suspects the local anatomy of the leak, and can adequately deal with the space—then this may be his golden moment for performing secondary open thoracotomy

If these considerations do not apply, and closed drainage has been carried out, an elective postresection thoracoplasty may be the only useful alternative

SUMMARY

An analysis is presented of 30 cases of postlobectomy space, occurring in a series of 135 lobectomies for tuberculosis Significant factors in the genesis of this complication are discussed Measures for both prophylaxis and postoperative management of the postresection apical pneumothorax are suggested

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LUNG COMPARTMENT DETERMINATION

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WITH the rapid growth of cardiopulmonary surgery there has arisen a great need for a method of determining just what is the true normal value of such factors as the cardiac index, total lung volume, residual capacity, vital capacity, maximum breathing ability, oxygen consumption and removal, etc.

The present methods of predicting the normal values of these function studies for any specific patient are based upon his body surface area, or are assumed to be some average value which has been determined upon a series of healthy subjects. It is hardly necessary to point out that body surface area is a very variable factor in itself, since it depends on the fat mass as well as weight and height. On the other hand, if one uses an "average value" as a predicted value for a specific patient that too is equally variable.

METHOD

The total lung capacity can be calculated from a single inspiratory posteroanterior chest film.

We had a series of clinically normal standing subjects inspire maximally from a recording spirometer and at the point of maximal inspiration an x-ray (posteroanterior) picture was taken. This was then immediately repeated in full expiration, after which it was done in the lateral plane at full inspiration and expiration while breathing into the spirometer. These last films were soon found to be unnecessary.

Residual air determinations were carried out after the roentgenology with consideration of the resting and fasting state. Our modification of the Cournand open-circuit method was used.[1]

CALCULATIONS

The whole thoracic cage is treated mathematically as a paraboloid of revolution with the vertex at the center of a horizontal line joining the highest points of the inner borders of the first ribs and "base" on the horizontal line joining the attachment of the diaphragm to the ribs in the midaxillary lines (Figs. 1 and 2).

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$$V = \frac{\pi}{2} ab^2 \qquad \text{(Ref. 2)}$$

Where V = volume of paraboloid of revolution of cage in milliliters.
a = distance from vertex to base in centimeters.
b = length of one-half "base" in centimeters.

To obtain the total lung air capacity one must subtract from this the volumes of the "diaphragmatic dome" above the "base line," the heart, the superior mediastinum and adjacent vertebral bodies, the posterior mediastinum and its adjacent vertebral bodies together with the lung parenchymal and blood volume.

The "diaphragmatic dome" is treated as the sum of two half segments of a sphere to allow for differences in the volumes of the right and left leaves (Figs. 1 and 3).

$$V = \frac{\pi}{6} h^2 (3b - h) \qquad \text{(Ref. 3)}$$

Where V = volume in milliliters of one-half segment of sphere.
b = length in centimeters of "one-half base."
h = height of top of diaphragmatic leaf from base line in centimeters.

This is calculated for each diaphragmatic leaf and the two added together to give the volume of the total diaphragmatic correction.

The heart is considered to be a paraboloid of revolution with vertex at the center of the base of the heart as soon "radiologically" where the heart borders become confluent with the superior mediastinum, usually at the upper border of the seventh rib posteriorly, or the lower border of the third rib anteriorly, and "base" on a horizontal line at the diaphragmatic surface of the heart (where the dome of the left leaf of the diaphragm and the inferior border of the heart merge) (Figs. 1 and 3).

The general formula for a paraboloid of revolution becomes:

$$V = \frac{\pi}{2} a_2 \frac{(t_1 - t_2)^2}{2}$$

Where V = volume of heart in milliliters.
a_2 = vertical distance from base of heart to level of diaphragmatic dome in centimeters.
t_1 = distance from midline to right border in centimeters.
t_2 = distance from midline to left border of heart in centimeters.

The superior mediastinum and adjacent vertebral bodies are treated as one unit mathematically. Since this is part and parcel of the original thoracic cage paraboloid of revolution, one may visualize it as a parabolic surface in cross section which when multiplied by the width "w" of the superior mediastinum will give its volume in milliliters. The base of this parabola is equal to the distance in centimeters from the midline, at the level of the upper border of the heart, laterally to the inside of the thoracic cage in the midaxillary line labeled b_1 on Figs. 1 and 2.

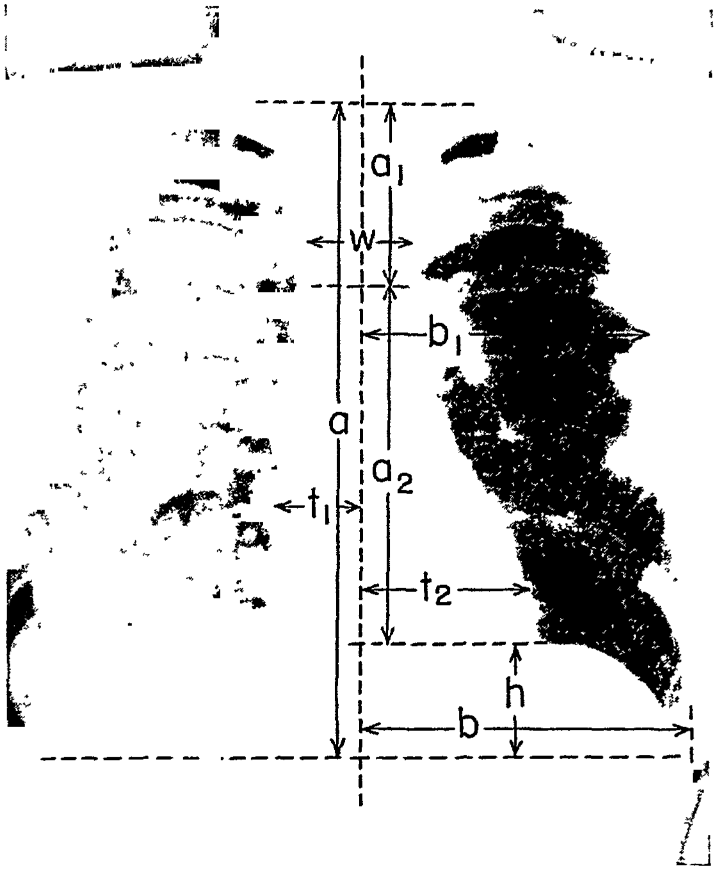


Fig. 1.

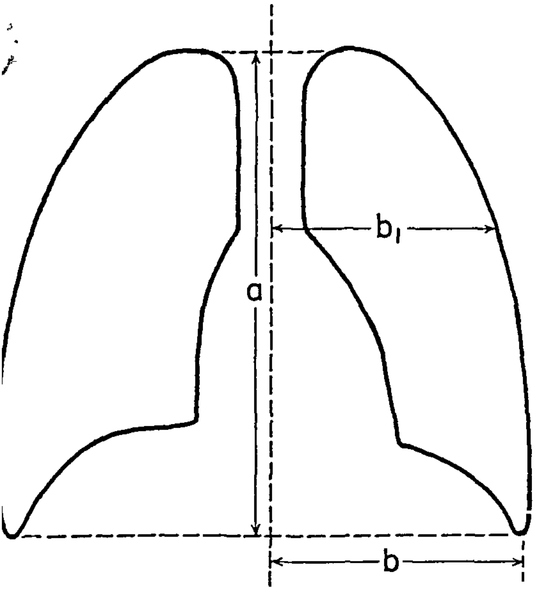


Fig. 2.

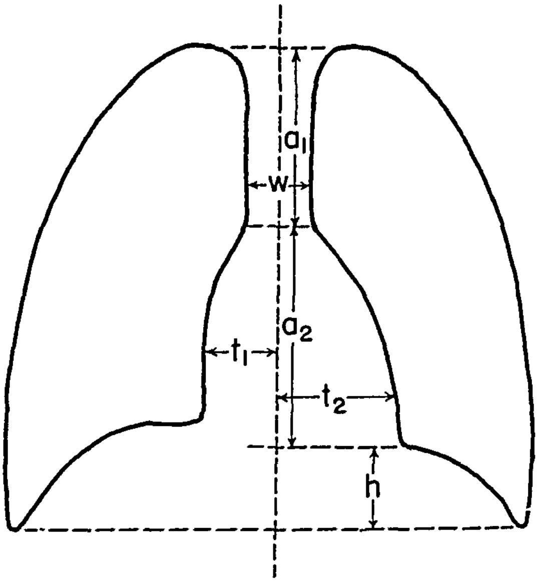


Fig. 3.

The general formula for the area of a section of a parabola is:

$$A = \frac{4}{3} a_1 b_1 \qquad \text{(Ref. 4)}$$

Where A = area in square centimeters of parabolic surface.
a_1 = distance in centimeters from thoracic cage vertex to upper border of heart.
b_1 = distance from mid-point of upper border of heart measured horizontally laterally to inside of ribs.

Now $V = A \cdot w$.
Where V = volume in milliliters of superior mediastinum and adjacent vertebral bodies.
A = area of parabolic cross section in square centimeters.
w = width of superior mediastinum in centimeters.

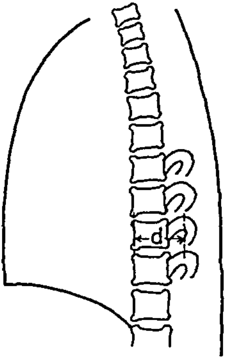


Fig. 4.

The volume of the vertebral bodies adjacent to the posterior mediastinum (which actually is the volume of the remaining vertebral bodies in the "thoracic cage") is obtained by multiplying their width by the length and depth of this column. The width is taken as equal to that of the superior mediastinum. The length is the distance from the upper border of the heart to the "base" of the thoracic cage ($a - a_1$) (Figs. 2 and 3) while the depth d is the distance from the anterior surface of the lower thoracic vertebral bodies to the inner surface of the angles of the ribs at the same level (Fig. 4). One may assume the depth to be 6.1 cm. since there is very little variation in this figure. With this assumption, a lateral film is unnecessary.

In correcting for the volume of the lung parenchyma and its contained blood, our pathology department has found this to vary from 685 ml. to 1,087 ml. We classify the lung volumes into Groups 1 to 5, respectively, 700, 800, 900, 1,000, and 1,100 ml. An average volume is 900 ml. From the x-ray film, one decides to which group the patient belongs and adds 100 ml. to this volume because the x-ray is taken at full inspiration when the output of the right ventricle temporarily is approximately 26 c.c. more than the left per beat so that the in vivo volume is approximately 200 c.c. more than the full expiratory

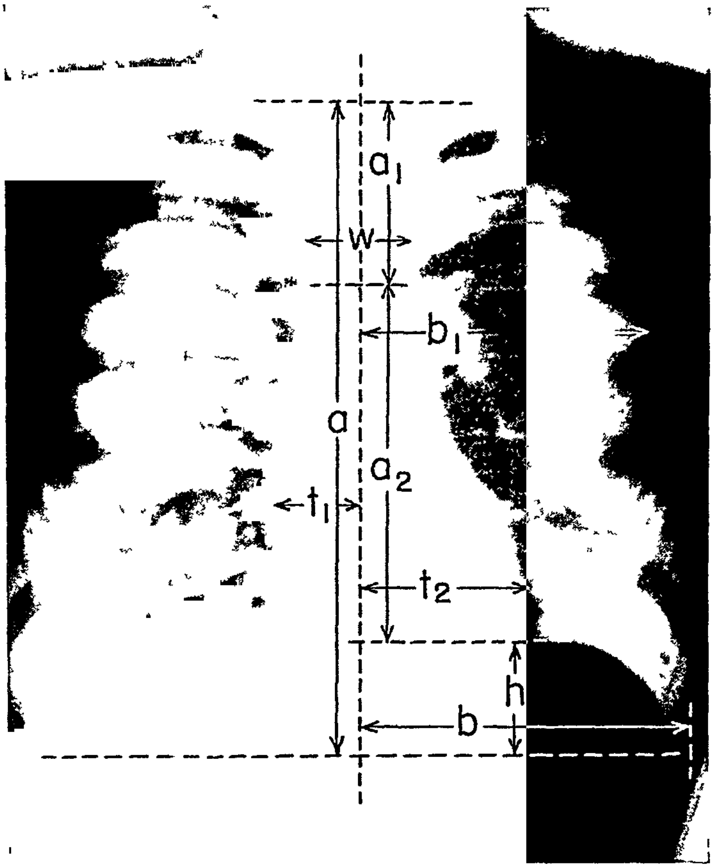


Fig. 1

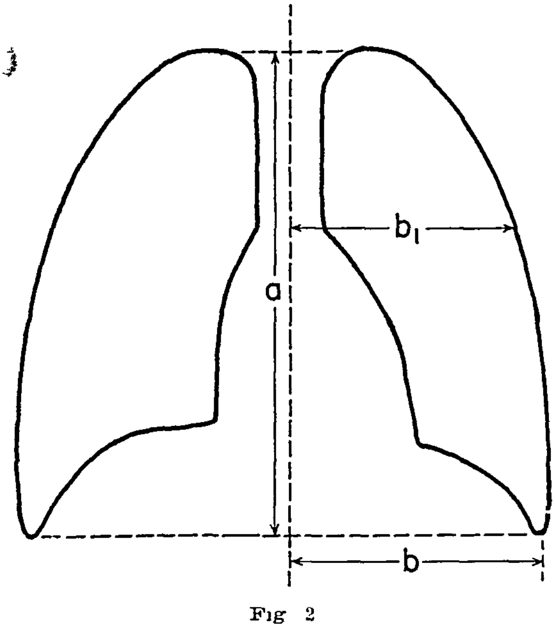
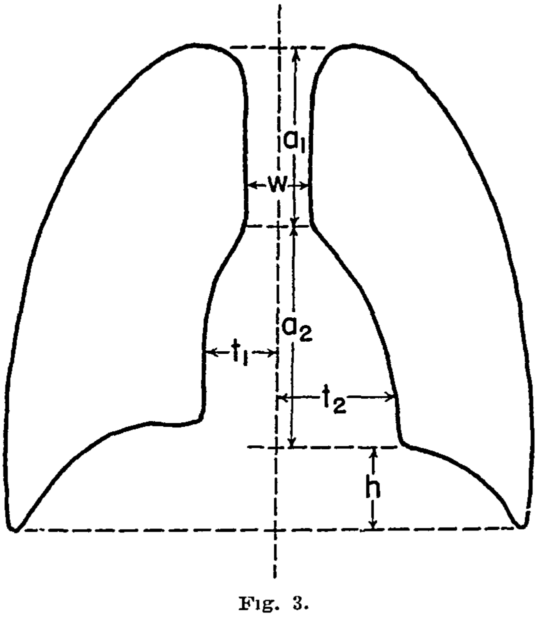


Fig 2



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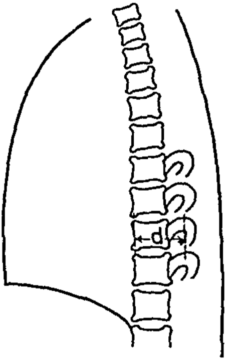

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TABLE I

PATIENT	AGE AND SEX		VITAL CAPACITY	RESIDUAL CAPACITY (MILLILITERS)	EXPERIMENTAL TOTAL CAPACITY (MILLILITERS)	CALCULATED TOTAL CAPACITY (MILLILITERS)	DIFFERENCE (MILLILITERS)	PER CENT DIFFERENCE
W. I.	41	F	4016	1935	5951	5928	− 23	0.39
G. A.	33	M	4740	940	5680	5720	+ 40	0.75
G. J.	40	M	5300	3320	8620	8557	− 63	0.73
H. R.	25	M	4107	1986	6093	6160	+ 67	1.07
C. J.	51	M	3510	2314	5825	5883	+ 58	0.97
K. G.	19	M	3668	1394	5062	5038	− 24	0.47
M. J.	41	M	4784	1697	6480	6400	− 80	1.18
C. M.	27	M	5276	1237	6513	6557	+ 44	0.67
M. W.	30	M	5480	1685	7165	7170	− 5	0.08
L. A.	31	M	6798	2134	8932	9030	+ 98	1.08
W. F.	47	F	1840	1028	2868	2888	+ 58	0.50
P. S.	28	M	3970	2278	6248	6221	− 27	0.43
R. H.	26	M	5000	1397	6397	6409	+ 12	0.05
W. A.	28	M	5650	1630	7280	7404	+124	1.70
G. H.	34	M	4900	1152	6052	6090	+ 38	0.63
J. H.	34	F	2890	871	3761	3766	− 05	0.01
C. J.	15	M	4620	775	5395	5314	− 89	1.60
S. J.	27	M	3950	1115	5065	5121	+ 56	1.10
R. T.	36	M	3840	1705	5545	5623	+ 78	0.71
M. R.	34	M	4150	910	5060	5070	+ 10	0.05
J. J.	24	M	3240	734	3974	3910	− 64	1.61
D. C.	23	M	5180	1110	6290	6340	+ 50	0.79

TABLE II

PATIENT	CAGE	DIAPHRAGM		SUPERIOR MEDIASTINUM	INFERIOR MEDIASTINUM	LUNG AND BLOOD	HEART
		RIGHT	LEFT				
W. I.	10880	1160	571	700	480	1000	1051
G. A.	12760	636	636	1350	696	1100	1300
G. J.	14860	1475	764	1005	797	1100	965
H. R.	10860	496	496	1045	586	1100	980
C. J.	9250	340	340	695	495	900	600
K. G.	9300	785	785	662	500	900	630
M. J.	11450	2205	900	504	667	900	775
C. M.	11720	1121	520	779	530	1100	1113
M. W.	12600	1275	797	714	555	1200	888
L. A.	15180	1035	923	900	697	1200	1395
W. F.	6120	548	112	465	375	900	821
P. S.	10300	608	410	876	403	1000	881
R. H.	10550	754	544	806	458	900	679
W. A.	13330	1437	579	975	590	1200	1145
G. H.	10420	814	468	732	472	1200	647
J. H.	5770	58	58	560	196	800	332
C. J.	9700	884	652	575	445	900	930
S. J.	11100	975	975	621	420	1000	1040
R. T.	10720	870	535	1022	655	1100	915
M. R.	9450	755	755	550	405	900	880
J. J.	7280	706	360	544	260	800	700
D. C.	11570	800	577	1040	595	1100	1120

RESULTS

The difference between the experimentally observed and calculated values for the total lung capacity was 0.75 per cent over a range or 0.01 per cent to 1.70 per cent in 22 subjects.

Table I is a summary of the results. Table II gives a summary of the components of the calculations used in their derivation.

DISCUSSION

These results show how accurately the total lung capacity can be calculated from the single posteroanterior film.

On our surgical service where all patients are studied in our cardiopulmonary laboratory we repeatedly found that the available predicted normal range was too wide to give us a proper estimate of a patient's deviation from his own normal. Our cardiac cases with grossly increased residual volume (unless they had very large hearts) and diminished vital capacity were especially hard to evaluate.

This method is applicable to all forms of chest pathology. We are now using it in our daily work.

SUMMARY

The vital capacity was determined on a recording spirometer. An x-ray film (posteroanterior) at full inspiration was taken simultaneously. The residual capacity was then determined. Clinically normal subjects were used throughout.

The total lung capacity was calculated by the method outlined from a single posteroanterior x-ray film in full inspiration.

There was an average difference of 0.7 per cent between the experimentally observed and the calculated total lung capacity.

The range of this difference was from 0.01 to 1.75 per cent.

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PATIENT	AGE AND SEX		VITAL CAPACITY	RESIDUAL CAPACITY (MILLILITERS)	EXPERIMENTAL TOTAL CAPACITY (MILLILITERS)	CALCULATED TOTAL CAPACITY (MILLILITERS)	DIFFERENCE (MILLILITERS)	PER CENT DIFFERENCE
W. I.	41	F	4016	1935	5951	5928	− 23	0.39
G. A.	33	M	4740	940	5680	5720	+ 40	0.75
G. J.	40	M	5300	3320	8620	8557	− 63	0.73
H. R.	25	M	4107	1986	6093	6160	+ 67	1.07
C. J.	51	M	3510	2314	5825	5883	+ 58	0.97
K. G.	19	M	3668	1394	5062	5038	− 24	0.47
M. J.	41	M	4784	1697	6480	6400	− 80	1.18
C. M.	27	M	5276	1237	6513	6557	+ 44	0.67
M. W.	30	M	5480	1685	7165	7170	− 5	0.08
L. A.	31	M	6798	2134	8932	9030	+ 98	1.08
W. F.	47	F	1840	1028	2868	2888	+ 58	0.50
P. S.	28	M	3970	2278	6248	6221	− 27	0.43
R. H.	26	M	5000	1397	6397	6409	+ 12	0.05
W. A.	28	M	5650	1630	7280	7404	+124	1.70
G. H.	34	M	4900	1152	6052	6090	+ 38	0.63
J. H.	34	F	2890	871	3761	3766	− 05	0.01
C. J.	15	M	4620	775	5395	5314	− 89	1.60
S. J.	27	M	3950	1115	5065	5121	+ 56	1.10
R. T.	36	M	3840	1705	5545	5623	+ 78	0.71
M. R.	34	M	4150	910	5060	5070	+ 10	0.05
J. J.	24	M	3240	734	3974	3910	− 64	1.61
D. C.	23	M	5180	1110	6290	6340	+ 50	0.79

TABLE II

PATIENT	CAGE	DIAPHRAGM		SUPERIOR MEDIASTINUM	INFERIOR MEDIASTINUM	LUNG AND BLOOD	HEART
		RIGHT	LEFT				
W. I.	10880	1160	571	700	480	1000	1051
G. A.	12760	636	636	1350	696	1100	1300
G. J.	14860	1475	764	1005	797	1100	965
H. R.	10860	496	496	1045	586	1100	980
C. J.	9250	340	340	695	495	900	600
K. G.	9300	785	785	662	500	900	630
M. J.	11450	2205	900	504	667	900	775
C. M.	11720	1121	520	779	530	1100	1113
M. W.	12600	1275	797	714	555	1200	888
L. A.	15180	1035	923	900	697	1200	1395
W. F.	6120	548	112	465	375	900	821
P. S.	10300	608	410	876	403	1000	881
R. H.	10550	754	544	806	458	900	679
W. A.	13330	1437	579	975	590	1200	1145
G. H.	*10420*	*814*	*468*	*732*	*472*	*1200*	*647*
J. H.	5770	58	58	560	196	800	332
C. J.	9700	884	652	575	445	900	930
S. J.	11100	975	975	621	420	1000	1040
R. T.	10720	870	535	1022	655	1100	915
M. R.	9450	755	755	550	405	900	880
J. J.	7280	706	360	544	260	800	700
D. C.	11570	800	577	1040	595	1100	1120

RESULTS

The difference between the experimentally observed and calculated values for the total lung capacity was 0 75 per cent over a range or 0 01 per cent to 1 70 per cent in 22 subjects

Table I is a summary of the results Table II gives a summary of the components of the calculations used in their derivation

DISCUSSION

These results show how accurately the total lung capacity can be calculated from the single posteroanterior film

On our surgical service where all patients are studied in our cardiopulmonary laboratory we repeatedly found that the available predicted normal range was too wide to give us a proper estimate of a patient's deviation from his own normal Our cardiac cases with grossly increased residual volume (unless they had very large hearts) and diminished vital capacity were especially hard to evaluate

This method is applicable to all forms of chest pathology We are now using it in our daily work

SUMMARY

The vital capacity was determined on a recording spirometer An x ray film (posteroanterior) at full inspiration was taken simultaneously The residual capacity was then determined Clinically normal subjects were used throughout

The total lung capacity was calculated by the method outlined from a single posteroanterior x ray film in full inspiration

There was an average difference of 0 7 per cent between the experimentally observed and the calculated total lung capacity

The range of this difference was from 0 01 to 1 75 per cent

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MEDIASTINAL TUMORS AND CYSTS

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THE present paper is a survey of primary mediastinal tumors and cysts, treated at the Clinic of Thoracic Surgery at Sabbatsberg Hospital during the period 1944 to 1954. As the survey is based on a patho-anatomic classification, we have included only those cases documented with patho-anatomic material.

The following lesions are excluded from this study: (1) carcinoma and benign tumors of the esophagus; (2) mediastinal extension or metastasis of bronchial carcinoma and other metastatic tumors as well; (3) intrathoracic goiter; and (4) systemic diseases and non-neoplastic processes such as leukemia, Hodgkin's disease, tuberculoma, sarcoidosis, aneurysms, and parasitic cysts.

Table I shows the composition of our material in all 155 cases. Some rare tumors and cysts, other than the processes just mentioned, are missing in the material: aberrant parathyroid adenoma and cysts of esophageal and enterogenous origin. We have found no specimens of these kinds among the patients treated at the Clinic in these eleven years. The absence of enterogenous cysts is explained by the fact that children usually are not seen in this Clinic.

Table I

Schwannoma and neurofibroma	38
Ganglioneuroma	11
Ganglioneuroblastoma	3
Neurogenic sarcoma	4
Sympathicoblastoma	2
Fibroma	2
Lipoma	1
Fibromyxosarcoma	5
Chondrosarcoma	1
Hemangiomatous tumors	5
Lymphangioma	7
Lymphosarcoma	5
Plasmacytosarcoma	1
Thymoma	19
Thymic cyst	1
Adult teratoma	14
Malignant teratoma	2
Tumor of bronchial adenoma type	3
Bronchial cysts	21
Pericardial cysts	6
Cysts of uncertain origin	4
Total	155

It will be observed, however, that we have taken up 3 cases classed as bronchial adenoma in our material. These were large tumors, wholly localized

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within the mediastinum and without any conspicious anatomic connection with lung, bronchi, or trachea. They presented the typical microscopic structure of bronchial adenoma. We have not been able to find any similar cases in the literature. We believe these tumors are related to the teratomas.

In order to determine whether the percentage distribution of the various diagnoses coincides with that found in other homogeneous series, we have made a comparison, as shown in Table II. In this Table, we have excluded from the compared series, all cases not included in our series. It will be seen that there is considerable statistical incongruity between the distributions in the different series. If they are summed up, however, the distribution in the combined material of 241 cases tallies rather well with that in ours. The similarity is good in some of the larger groups, i.e., benign neurogenic tumors and bronchiogenic cysts. Teratomas, on the other hand, are less frequent in our series than the combined material, partly due to the astonishingly high frequency of these specimens in the material of Curreri and Gale.[11] Thymoma is more frequent in our material. We have rather few pericardial cysts but rather many lymphangiomas. It is possible that some pericardial cysts in the earlier authors' materials in reality have been lymphangiomas. These two cystic affections are rather difficult to separate.

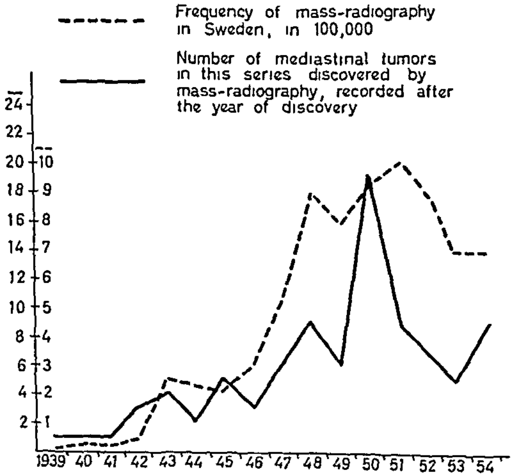


Fig. 1.—Annual distribution of mediastinal tumors discovered by mass radiography in this series compared with the frequency of mass radiography in Sweden.

Before we proceed with a description of the different groups of material, we want to stress the importance of mass radiography for the discovery of these mediastinal affections. It is, indeed, reasonable to say that the extensive use of mass radiography on the Swedish population during the last decade has been responsible for detection of most of this material. In [illegible] f our 155 cases (about 61 per cent), the mediastinal affection

TABLE II

TYPE	BLADES[7]	BRADFORD, MAHON, AND GROW,[9] 1947	BREWER AND DOLLEY,[10] 1949	CONKLIN,[13] 1950	CURRERI AND GALE,[11] 1949	SHIDLER AND HOLMAN,[49] 1952	TOTAL	PER CENT OF TOTAL	OUR SERIES	PER CENT OF OUR SERIES
Benign neurogenic	29	6	16	5	6	15	77	32.0	52	33.5
Malignant neurogenic	1	0	1	0	1	0	3	1.2	6	3.9
Fibroma	1	0	0	2	0	1	4	1.6	2	1.3
Lipoma	3	4	0	0	1	0	8	3.3	1	0.6
Connective tissue sarcomas	0	0	4	1	0	0	5	2.1	6	3.9
Hemangioma	0	0	0	0	0	0	0	0.0	5	3.2
Lymphangioma	0	1	1	1	1	0	4	1.6	7	4.5
Lymphosarcoma	2	0	0	4	1	0	7	2.9	5	3.2
Plasmacytosarcoma	0	0	0	0	0	0	0	0.0	1	0.6
Thymoma	6	3	2	0	2	4	17	7.5	19	12.3
Thymic cyst	0	0	0	0	0	0	0	0.0	1	0.6
Esophageal cysts	0	0	0	0	1	0	1	0.2	0	0.0
Pericardial cysts	10	7	2	2	2	2	25	10.4	6	3.9
Bronchiogenic cysts	23	6	3	4	2	2	40	16.7	21	13.6
Bronchial adenoma	0	0	0	0	0	0	0	0.0	3	2.0
Benign teratoma	14	4	8	5	11	1	43	17.8	14	9.0
Malignant teratoma	6	0	0	0	1	0	7	2.9	2	1.3
Undefined cysts	0	0	0	0	0	0	0	0.0	4	2.6
Total	95	31	37	24	29	25	241	100.0	155	100.0

routine mass radiography. The percentage is about 68 for the benign and 23 for the malignant growths. Fig. 1 gives the distribution of the cases discovered by mass radiography in Sweden as to the year of discovery (full drawn line), and the frequency of mass radiography in Sweden during these years (in hundred thousands, dotted line). A rather close agreement can be noted between the frequency of mass radiography on the one hand and the number of cases discovered by this method, on the other hand.

The specific sites of origin of the various mediastinal tumors and cysts in our series are shown in Table III. There is a definite similarity between the dispersion of the different kinds of tumors in our material and the surveys of localization given by Schlumberger,[44] and Hochberg and associates, among others.

TABLE III

Anterior Superior Mediastinum —		*Posterior Superior Mediastinum*	
Thymoma	16	Schwannoma and neurofibroma	24
Adult teratoma	10	Ganglioneuroma	6
Malignant teratoma	2	Malignant neurogenic tumors	2
Lymphosarcoma	5	Bronchial cysts	6
Fibroma	2	Adult teratoma	1
Lipoma	1	Hemangioma	1
Fibrosarcoma	1	Plasmacytosarcoma	1
Hemangioma	4	Chondrosarcoma	1
Lymphangioma	2	Total	42
Bronchial adenoma	2		
Bronchial cysts	4	*Posterior Inferior Mediastinum —*	
Thymic cysts	1	Schwannoma	14
Undefined cysts	4	Ganglioneuroma	8
Total	54	Malignant neurogenic tumors	4
		Fibrosarcoma	3
Hilar Region —		Lymphangioma	4
Bronchial cysts	6	Bronchial cysts	5
Adult teratoma	3	Bronchial adenoma	1
Thymoma	3	Total	39
Lymphangioma	1		
Total	13		
Anterior Inferior Mediastinum —			
Fibrosarcoma	1		
Pericardial cysts	6		
Total	7		

NEUROGENIC TUMORS

These are the most frequent among the mediastinal tumors. According to Schlumberger,[44] reports of at least 230 cases have been published up to 1951, Hirschfeld,[illegible] however, gives the figure as 172 cases reported up to the same year.

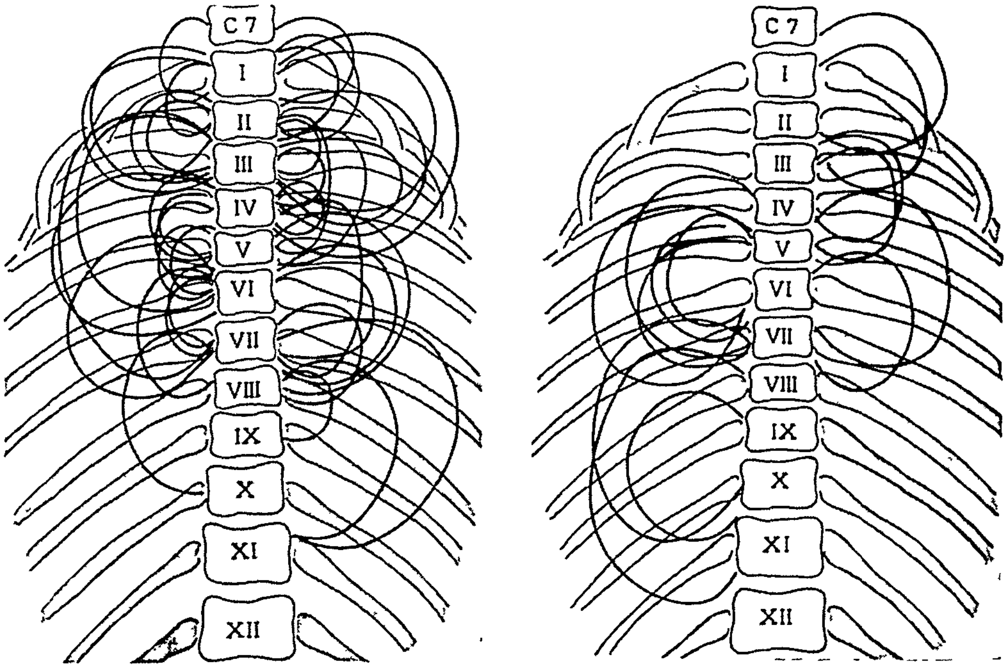
Histologically, they are classed as benign tumors: schwannoma (neurinoma), neurofibroma, and ganglioneuroma and malignant tumors: neurogenic sarcoma (malignant schwannoma) and sympathicoblastoma. There is also a tumor called ganglioneuroblastoma, which microscopically, seems to be intermediate between a ganglioneuroma and a sympathicoblastoma.

Of these tumors, schwannoma and neurofibroma are the most common, followed by ganglioneuroma. According to Rodriguez and co-workers,[illegible] 90 cases of mediastinal ganglioneuroma had been published up to 1951.

According to Ackerman and Taylor,[1] 14.6 per cent of all mediastinal neurogenic tumors are malignant, but Brewer and Dolley[10] give a figure as high as 33 per cent. Within the malignant groups, malignant schwannoma seems to be most frequent. Hollingsworth[28] (1946), however, collected 18 cases of mediastinal sympathicoblastoma from the literature and added 1 of his own, Davis[15] (1947) found 14 cases in the literature and presented 2 of his own cases.

A comparison of the frequency in Ackerman and Taylor's[1] homogeneous material of 48 mediastinal neurogenous tumors, and in ours of 58 cases, follows:

	ACKERMAN AND TAYLOR	OURS
Neurinoma and neurofibroma	27	38
Ganglioneuroma	7	11
Ganglioneuroblastoma	7	3
Neurogenic sarcoma	4	4
Sympathicoblastoma	3	2
	48	58

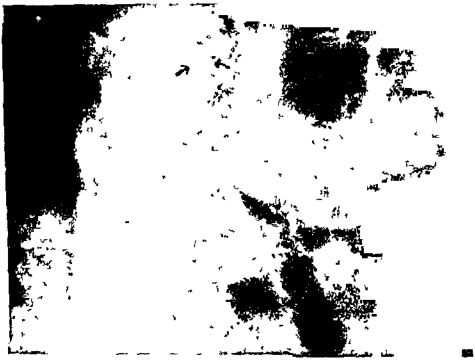


A. *B.*

Fig. 2.—Location and approximate size of: *A*, 38 neurinomas and neurofibromas, and *B*, of 14 ganglioneuromas and ganglioneuroblastomas.

As seen, the proportion of clearly malignant tumors is lower in our series than in Ackerman and Taylor's material (10.3 per cent to 14.6 per cent) and the proportion of ganglioneuroblastomas is lower in ours also. This is probably due to the fact that the average age in these tumor groups is low, as children are infrequently seen in our Clinic.

The *benign neurogenic tumors* (including ganglioneuroblastomas) are all similar as to site and gross anatomy. They are invariably located at the costovertebral sulcus, growing close to the lateral surface of the columna and the most proximal part of the ribs. They arise subpleurally and bulge into the thoracic cavity with a pleural covering. They are always well encapsulated. The location in this series varies from the level of the seventh cervical vertebra *to a point just above the diaphragm, i.e.,* the level of the tenth thoracic vertebra (Fig. 2). The distribution on both sides is about even (24 cases on the left side, 27 cases on the right side, and 1 case was bilateral). The size varies from that of a walnut to that of a large grapefruit. They are spherical or ovoid in shape with a smooth pleural surface. Their tissue is mostly solid, sometimes with degenerative cysts, grayish-white often yellow-spotted. The surgeons often report these tumors as being connected with the sympathetic cord and/or the spinal nerves (25 per cent and 63 per cent, respectively). In about 20 per cent of the cases, the tumor tissue shows an extension into one or several intervertebral foramina. The x-ray picture of dilated foramina is an important diagnostic sign in these cases (Fig. 3)!

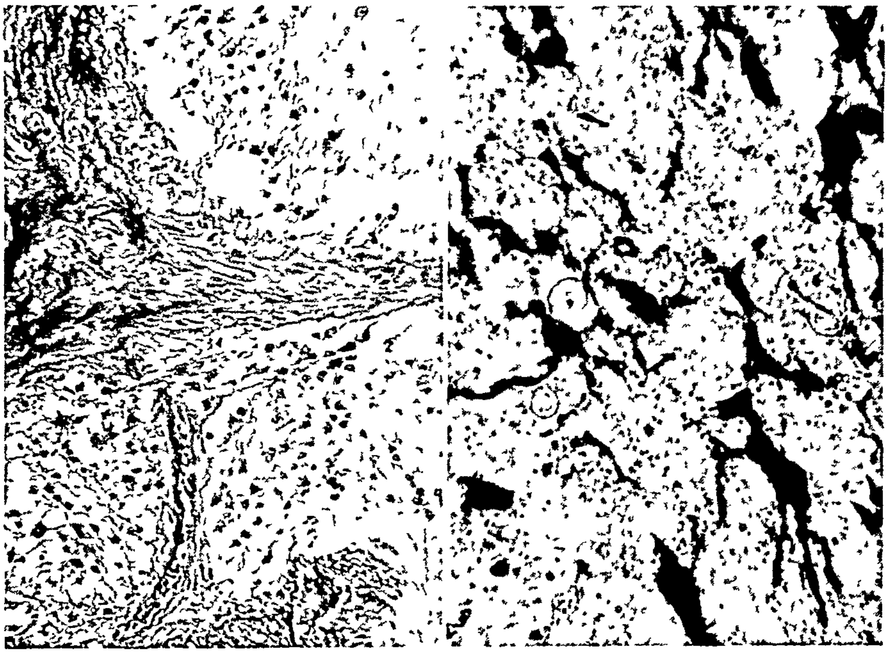


of a dilated intervertebral foramen in a case of neurinoma with
e area designated by the two arrows shows the absence of normal

In our series, the tumor has presented the gross picture just described whether the microscopic picture was that of schwannoma, combined schwannoma-neurofibroma, ganglioneuroma, or ganglioneuroblastoma. In only 2 cases, with a pure neurofibromatous histologic picture, has the gross picture been atypical; sausage-shaped, markedly gelatinous tumors. In 1 of these cases, the tumor was a local manifestation of a generalized von Recklinghausen's disease. Multiple small nodules along the intercostal nerves were additionally observed.

The histologic picture in 36 of these tumors has been that of schwannoma, either pure, or in a varying degree combined with neurofibromatous structures. As many of the tumors present regressive changes with abundant formation of hyaline fibrous tissue, we find it difficult to draw the border line between schwannoma and neurofibroma. However, in another 2 cases, the picture was that of a pure neurofibroma without appreciable schwannoma-pattern and also without regressive changes. These 2 cases presented an atypical gross picture, as described before.

Eleven of the tumors were mature ganglioneuromas and 3 were tumors with a less mature structure namely, ganglioneuroblastomas. The dominating tissue in all these neoplasms is neurofibromatous, most often consisting of a network of bundles separated by a loose connective tissue. The bundles contain Schwann cells, rests of myelin sheets, and sometimes demonstrable axons. Nerve cells appear scattered or in small nests. In the ganglioneuromas, the nerve cells are of a mature type and often sparse. The ganglioneuroblastomas are rich in nerve cells of an immature type, varying in size, often multinucleated. The nests of these cells are surrounded by a peculiar loose, cobwebby tissue, rather similar to a glial network. Some of the fibers in this tissue seem to be axons. With a combined gold-silver-impregnation method, we have also been able to demonstrate glia cells often of an immature uni- or bipolar type in these islands (Fig. 4).



A. *B*

Fig 4.—Ganglioneuroblastoma *A*, Nests of nerve cells surrounded by a cobwebby tissue (van Gieson stain, ×120) *B*, Glia cells in the cobwebby tissue (Silver-gold impregnation, ×560)

The incidence of females, in our series and Ackerman and Taylor's[1] series of benign neurogenic tumors and ganglioneuroblastoma, is the same, 60 per cent. The age distribution is different for the various histologic groups Thus, for the schwannoma-neurofibroma group the mean age at operation is 43, for ganglioneuromas, 25, and for ganglioneuroblastoma 6 years and 7 months. The corresponding figures in Ackerman and Taylor's[1] series are 43, 14, and 6 years, 5 months, respectively.

These benign tumors grow slowly and often attain a considerable size without giving any symptoms. Thus, 36 of the 52 patients in this series had been quite free from symptoms and the tumor was accidentally discovered by mass radiography. Another 10 cases were also diagnosed in this way although the patients had symptoms which did not cause a medical consultation. The symptoms were mostly a feeling of pressure, slight pain, and dyspnea. In 4 cases, with the tumor located at the level of the seventh cervical to the third thoracic vertebra, there was a well developed Horner's syndrome. In 2 cases, one neurofibroma and one ganglioneuroma, the patient showed signs of von Recklinghausen's disease.

All of the patients underwent surgery.

Of the 52 patients, 49 are living three months to eleven years after the operation. Two died postoperatively (of bronchopneumonia and myocardial infarction, respectively). One died seven years after operation of cardiac disease. All the survivors, except 3, are quite free from symptoms. One patient complains of slight thoracic pains, one of slight discomfort in the hand on the side operated upon and in 1 case, the Horner syndrome has persisted. It is of interest that our 3 patients with ganglioneuroblastoma are asymptomatic 3 years, 9 months, 2 years, 3 months, and 6 months after operation, respectively. This fact has caused us to describe the ganglioneuroblastomas together with the benign tumors. In Ackerman and Taylor's[5] series of 7 cases in this group, 2 died because of metastases.

The malignant neurogenic tumors in this series are 6, i.e., 2, sympathicoblastomas, 3, sarcomas with recognizable schwannoma structures, and 1, sarcomatous mediastinal neurofibroma in a case of von Recklinghausen's disease. The sympathicoblastomas occurred in males, aged 15 and 8, respectively. The first patient was operated upon at the age of 5 for an extradural spinal tumor of suspected neuroblastoma type and ten years later presented a rapidly growing and infiltrating inoperable growth at the same level as the spinal tumor. A biopsy showed a clear sympathicoblastoma, and the boy died ten months later from metastases. The other patient had an orange sized infiltrating tumor in the costovertebral sulcus. This patient died from cerebral metastases two months after the operation.

The three sarcomas of schwannoma type occurred in 1 man and 2 women aged 30, 31, and 51 years, respectively. The tumors were all located in the costovertebral sulcus, an orange to a cocoanut in size. It is interesting that none of them showed infiltrative growth grossly, and the surgeon reported complete removal. However, 2 of the patients died of the tumor within two years and one is living with recurrence one and one half years after operation. One of these patients had generalized von Recklinghausen's disease.

The last patient with malignant neurogenous tumor, a woman 25 years of age, had suffered from von Recklinghausen's disease since childhood. She displayed a rapidly growing pear shaped, seemingly well delimited neoplasm in the costovertebral angle and had, in addition, multiple small tumors along the costal nerves. In spite of complete operation, she died with local recurrence and metastases eight months later.

CONNECTIVE TISSUE TUMORS

Fibromas are comparatively rare in the mediastinum. According to Blades,[6] 32 cases had been reported up to 1940, a number of these probabl being fibrosarcomas. In the series included in Table II there are 4 cases of fibroma. Mediastinal *myxoma* seems to be very rare.

In our series, there are 2 cases of fibroma, both located in the upper anterior mediastinum, occurring in women aged 23 and 48 years, respectively. One of these growths, though giving rise only to slight asthmatic troubles, had attained a size larger than a child's head. Both patients are alive and well four years after the operation. We have no case of mediastinal myxoma.

Fibrosarcomas are also rare in the mediastinum. Heuer and Andrus[25] (1940) collected 17 cases from the literature, which were mostly located in the posterior mediastinum. Brewer and Dolley[10] reported 4 cases of their own.

In our series, we have 5 cases, 3 in women and 2 in men, with ages ranging from 23 to 66. The size of the tumors varied between that of an orange and a large grapefruit. In 2 cases, the location was anterior; in 3, posterior-inferior, one of the latter tumors extending into the retroperitoneal region. In 2 of the cases, the tumor was found to be well encapsulated. The preoperative symptoms, in 4 of the cases (less than one year's duration) were characterized by pains, cough, oppression, and dyspnea. In only 2 cases could a complete excision be performed. One of these patients died postoperatively (pulmonary embolism), the other died from metastasis four years after the operation. Of the other 3 patients 1 died shortly after the exploratory operation. Two others are alive, 1 of them two months and the other two years postoperatively, the latter with signs of recurrence.

Tumors classed as *fibroxanthoma* are often reported as being located in the mediastinum, in locations similar to those of the neurogenic tumors. It seems probable that most of them in reality were schwannomas or neurofibromas with xanthomatous degeneration. We observed this type of degeneration in 12 of our 38 tumor cases in the schwannoma-neurofibroma group. On the other hand, we have no tumor in this series which we have found reason to class as fibroxanthoma.

Lipomas are often reported in an intrathoracic position. Thus, Keeley and associates[29] (1953) found 57 cases in the literature and added 1 case. However, 10 of them were "hour-glass" tumors, protruding subcutaneously through intercostal spaces, and 6 were "cervicomediastinal." Of the remainder, at least 10, including the case of these authors, had a mid-line, clearly mediastinal location.

In the material of Sabbatsberg hospital, there are a number of lateral or supradiaphragmatic intrathoracic lipomas. Only 1 such tumor was located in the mediastinum, this one occurred in a man, 28 years of age, and showed a double grapefruit-sized lobulated, well-encapsulated tumor in the thymic region. Microscopically, it was a lipoma of mature type with small islands of remaining normal thymic tissue dispersed between the tumor nodules. The tumor did not give any symptoms and had been discovered by mass radiography six years before the operation. The patient is well six months after the operation.

A few cases of mediastinal *liposarcoma* have been reported in the literature, the majority of them with a benign clinical course, however. We have no such specimen in our series.

Chondromas and *chondrosarcomas* with mediastinal location arise in the vertebral structures, the head of the ribs, and perhaps, also, in the sternum and costal cartilages, and grow into the posterior or anterior mediastinum. About a dozen cases have been reported in the literature. Only 1 of these seems to have been a clear chondrosarcoma (Edwards[18]).

In this series, we have 1 case of chondrosarcoma, occurring in a man, 35 years of age, with a preoperative history of only four months with cough and thoracic pains. He had a double grapefruit-sized tumor in the posterior mediastinum, probably arising in the third thoracic vertebra. Two years after incomplete surgical excision, he returned to the Clinic with an inoperable recurrence and died with one year from metastases. Histologically, we found a multinodulated, bone-infiltrating tumor tissue, mostly of a cellular, immature type, similar to embryonal cartilage, but in places of a more mature chondroma type.

MYOGENOUS TUMORS

A few cases of mediastinal *leiomyoma* have been reported in the literature. Most of them have been located posteriorly and some of them may have arisen from esophageal musculature. We have no case of leiomyoma in our series.

Rhabdomyoma is occasionally seen as a tumor of the chest wall, but no case of pure rhabdomyoma with mediastnal location is known. The case of Fox and Hospers[19] was an anterior mediastinal teratoma containing rhabdomyosarcomatous tissue. We have no such tumor or any pure rhabdomyoma in our series.

ANGIOMAS

Hemangiomas are very rarely found in the mediastinum. Seybold and coworkers[47] (1949) reported 14 cases from the literature and added 3 of their own. Of these 17 cases, 8 showed signs of malignancy. During recent years, at least 3 more cases have been described, all benign (Schlumberger,[45] Thomas and associates,[51] and Grimes and associates.[22]) Schlumberger[45] stresses the fact that mediastinal teratomas may be partially hemangiomatous. Brindley[11] (1949) reported one tumor claimed to be a mediastinal glomangioma.

The present series includes four hemangiomatous tumors and one peculiar specimen, which perhaps is a malformation rather than a tumor.

The four hemangiomatous tumors were all located in the upper anterior mediastinum; walnut to orange in size. None of the tumors were encapsulated. All showed an infiltrative growth around the upper mediastinal vessels and in 1 case infiltration into the heart and lung also. Histologically, they all presented a rather peculiar picture. Two of the tumors were of identical appearance, both benign. In the third, the microscopic picture showed malignancy, but the patient has remained well ten years after operation. In the fourth case, the tumor was an infiltratively growing *hemangiopericytoma*; this patient died postoperatively.

In the fifth case, a well-encapsulated walnut-sized tumor was located in the costovertebral angle and was built up by cavernous blood-filled spaces interspersed with a heavily melanin-pigmented cellular tissue.

As all of these cases are instances of rare and morphologically peculiar specimens, they will be reviewed here briefly.

CASE REPORTS

CASE 1.—A woman, aged 29, had a history of pains and a pulsating sensation in the left arm of one year's duration. A firm tumor was palpable medially above the left clavicle. At operation, a walnut-sized, firm, grayish-red tumor was found growing as a cuff around the innominate vein and the adjacent part of the subclavian vein, moderately stenosing these vessels. It was extirpated together with the infiltrated parts of the veins. The patient is quite well three and one-half years after the operation. *Histology:* The tumor tissue had a more or less abundant fibrous stroma enclosing numerous small strands of cells, rather rich in cytoplasm and forming many syncytia (Fig. 5). The cells were arranged around narrow spaces, some empty, some containing red corpuscles. There was neither nuclear polymorphism, nor mitoses.

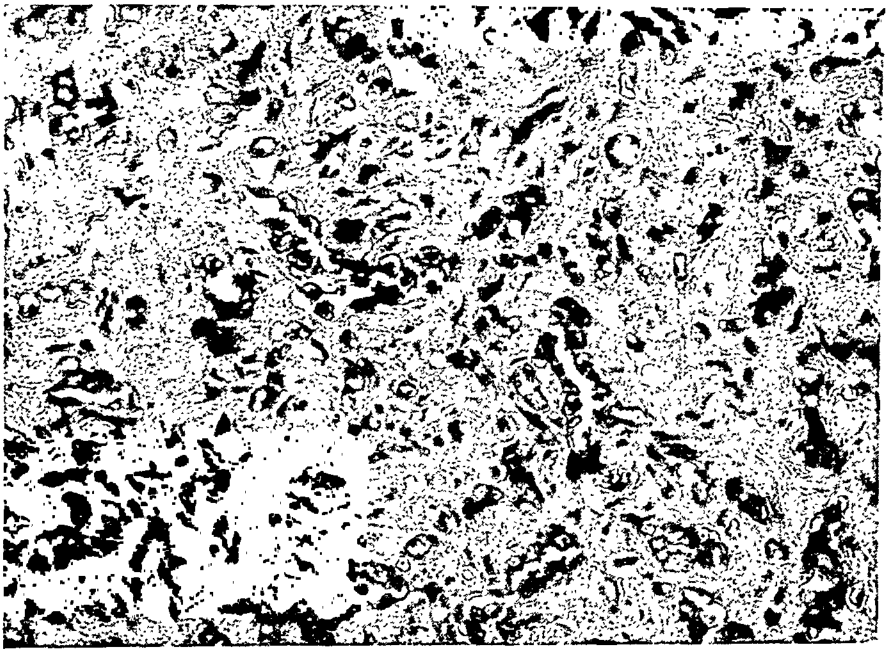


Fig. 5.—Case 1. Capillary hemangioma with syncytial endothelium cells. (van Gieson stain, ×430.)

CASE 2.—A man, aged 76, who had a history of uncertain duration of oppression, pains, dyspnea, swallowing troubles, and hoarseness. X-ray showed an upper mediastinal tumor with dislocation of the trachea and esophagus to the right. At thoracotomy a firm, orange-sized neoplasm was found in the upper anterior mediastinum, free from the lung, but obviously infiltrating around the big vessels. A frozen-section biopsy was erroneously interpreted as cancer and no attempt at surgical removal was made. After subsequent x-ray therapy, he was subjectively improved three months postoperatively. *Histology:* The same as in Case 1.

CASE 3.—A woman, aged 32, had a history of fatigue, raised temperature, cough, and tachycardia of 20 months' duration. Thoracotomy revealed an upper mediastinal neoplasm of grapefruit size, growing around the aorta ascendens and infiltrating through the pericardium, the wall of the left auricle, and the region of the left coronary sulcus (Fig. 6). The neoplasm was adherent to the lung and infiltrated the left upper lobe in one place. It was solid, grayish red, vascular, with necrotic patches. The patient died, postoperatively, after a partial removal. *Histology:* It was a cellular tumor with abundant narrow blood spaces,

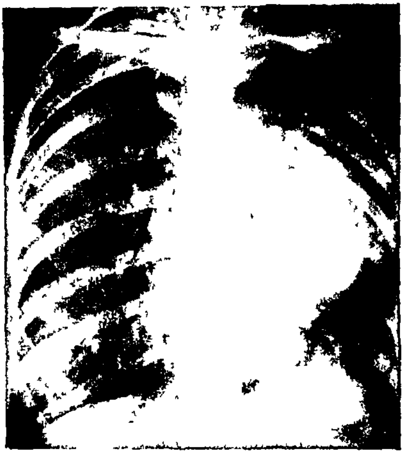


Fig 6 —Case 3 Roentgenogram of a mediastinal hemangiopericytoma

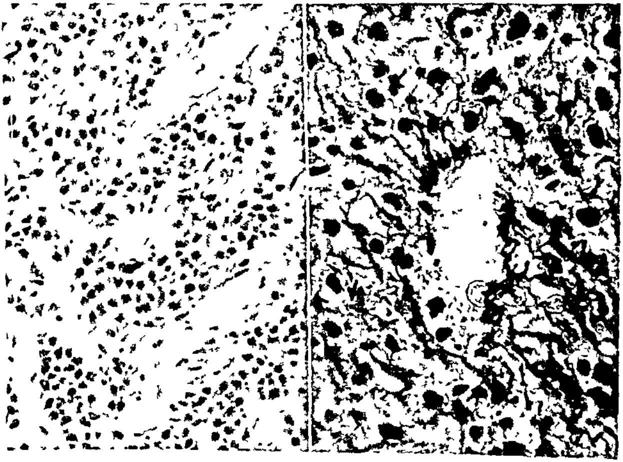


Fig 7 —Case 3 Hemangiopericytoma (*A*, van Gieson ×230 *B*, silver impregnation according to Laidlaw, ×560)

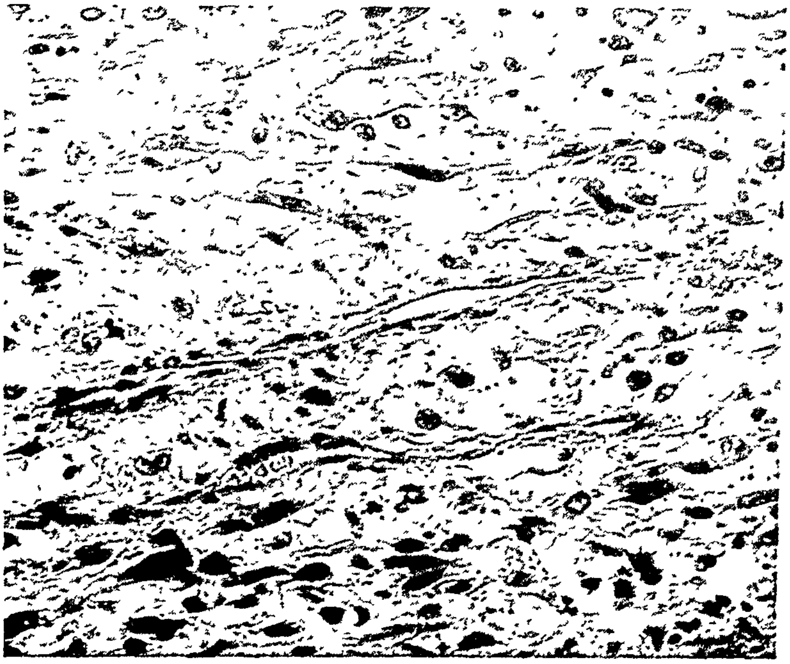


Fig. 8.—Case 4. Sarcomatous hemangioma. Strands of syncytial tumor cells imitating capillaries. (van Gieson stain, ×280.)

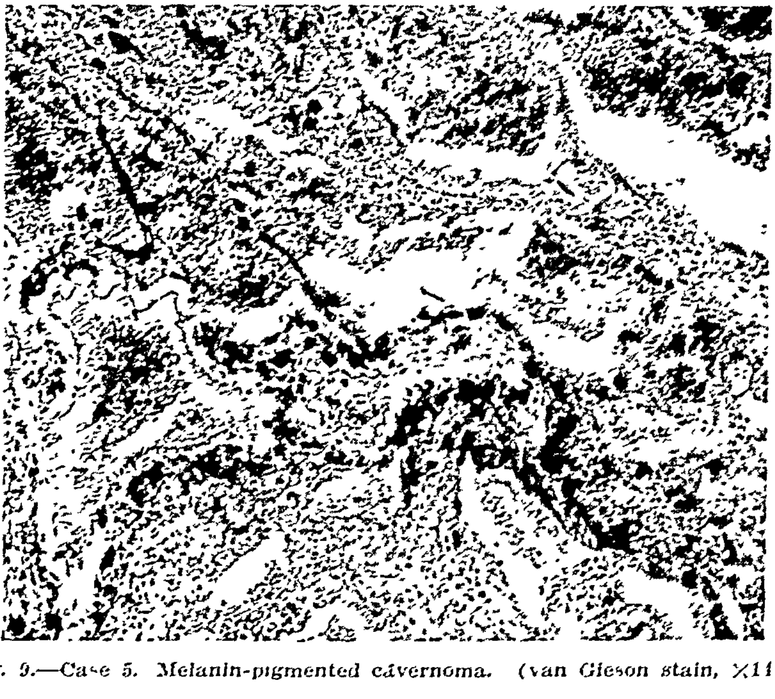


Fig. 9.—Case 5. Melanin-pigmented cavernoma. (van Gieson stain, ×110.)

lined with endothelial cells (Fig. 7). Peripheral to the lining cells were numerous polyhedral cells, rather rich in cytoplasm and with a moderate nuclear pleomorphism. There was a well-developed intracellular reticulum with a clear arrangement around the capillary spaces, which corresponded to the tumor called hemangiopericytoma, described by Stout[50] (1949).

CASE 4.—A man, aged 41, had an upper mediastinal tumor discovered by x-ray in connection with an acute respiratory infection. It grew slowly and symptomless during one year, giving slight thoracic pains only 6 weeks before operation. Thoracotomy revealed an orange-sized upper anterior mediastinal tumor growing infiltratively around the aortic arch and other big vessels and also infiltrating the pericardium. The cut surface was grayish with necrotic spots. The surgeon was able to remove the neoplasm in radical fashion. The patient is alive and well more than ten years after the operation. *Histology:* In the central parts, a clearly sarcomatous cellular tissue with bundles of elongated pleomorphic, mitosis-containing cells surrounding numerous small capillary spaces was seen. In the periphery, the tumor cells grew in long, often bifurcated strands in a loose connective tissue. The strands were built up by two layers of syncytial cells enclosing narrow spaces with a few red corpuscles, thus imitating young, newly formed capillaries (Fig. 8). It was classed as hemangiosarcoma.

CASE 5.—A woman, aged 55, had had coughing spells for 15 years. Six years before the operation, a mediastinal tumor was discovered by x-ray. Tests did not reveal any increase in size, and no new symptoms appeared. Thoracotomy revealed a walnut-sized well-encapsulated tumor in the right costovertebral sulcus at the level of the fourth thoracic vertebra. It seemed to be connected with the intercostal nerve, but had no intraforaminal extension. It was bluish in color with multiple blood-filled cystic spaces. After radical extirpation, the patient has remained well for more than two years. *Histology:* The tissue contained numerous cavernous blood-filled spaces with a thin endothelial covering. Between the spaces, there was a cellular tissue with small monomorphous, round nuclei and indistinct cell borders. The cytoplasm contained abundant fine or coarse granules of darkish-brown pigment, which presented a positive reaction to Masson's melanin stain. There was also much iron-positive pigment in places, probably hemosiderin. The cellular tissue revealed no structures similar to Schwannoma or neurofibroma and no nerve cells were to be found. We were not able to classify this specimen or to find any of similar description in the literature. We believe that it may possibly be a hamartoma of combined vascular and neurogenic origin (Fig. 9.).

Lymphangiomas in the mediastinum seem to be slightly more common than hemangiomas. According to Schlumberger,[44] 12 cases had been recorded up to 1948. In the series of Brewer and Dolley[10] (1949), Curreri and Gale[11] (1949), and Conklin[13] (1950), there are 3 more cases and Lob[36] has published 1 case (1951). Thus, at least 16 cases are known. The tumors have occurred predominantly in adults in an anterior location and have been mostly without symptoms.

In this series, we have 7 cases of lymphangioma (3 in men and 4 in women). Two of them were children (3, and 6 years of age); the others adults (whose ages ranged between 36 and 48 years). In our series, the anterior location does not predominate. We had 2 situated upper-anteriorly, 1 anteriorly in the hilar region, and 1 on the left side of the pericardial sac. On the other hand, we have 3 cases with lower posterior location, the tumor growing paravertebrally and supradiaphragmatically behind the heart, adherent to the pericardium. The smallest specimens have been about the size of an orange, the largest one covered the left side of the pericardium and had an extension of about 20 by 30 cm, but was not very thick. All the tumors were multicystic, either with a honeycombed cut surface throughout or with one main cyst. In 2 cases there were small extra cysts, separated from the main tumor. The cysts contained a water-clear or yellow-brownish fluid. Microscopically, the picture was characteristic: cystic spaces with an endothelial covering and thin fibrous wall, and in every case the specimen contained scattered streaks of smooth musculature. One may also find sparse lymphocyte infiltrates. The stroma tissue between the cysts always contained adipose tissue. In the 2 cases with upper anterior location, we did not find any thymus tissue in the stroma.

There is always some difficulty in differentiating mediastinal lymphangioma from thymic cyst and pericardial cyst. In our opinion, a thymic cyst seldom has smooth musculature in the wall and, if one had adequate material, one will always find rests of thymus tissue with Hassall's corpuscles adjacent to the cyst wall. The pericardial cysts do not show any smooth musculature, and the lining cells are (at least sometimes) cuboidal and not of the flat, endothelial type.

These lymphangiomas grow slowly and are symptomless. In all cases but one in our series, the tumor was discovered accidentally, either by mass radiography or by x-ray examination in connection with an acute respiratory infection. In the remaining case, there had been thoracic pains, dyspnea, and attacks of suffocation. In all the cases, it was possible to remove the tumor radically and all the patients are alive and well one and one-half to ten years after the operation.

LYMPHOSARCOMA AND PLASMACYTOSARCOMA

Malignant lymphoma, including Hodgkin's disease and leukemia, often produces mediastinal masses, especially in the upper anterior part of the mediastinum. Thus, patients are often operated upon suspected of having mediastinal tumors. In quite a few such cases, an erroneous histologic diagnosis of malignant thymoma is given. We have seen many instances of this in the material from this Clinic.

The lymphosarcomas do not constitute a very large group among the mediastinal neoplasms. In the series recorded in Table II, they averaged less than 3 per cent of all mediastinal tumors and cysts.

Plasmacytoma and plasmacytosarcoma seem to be extremely rare in the mediastinum. We have found only 1 case of the former (Childress and Adie[12]), and 1 of the latter (Divis[16]) reported in the literature.

In the present series, we have (with exclusion of Hodgkin's disease and leukemia) taken up 5 cases of mediastinal lymphosarcoma and reticulum-cell sarcoma, and 1 case of plasmacytosarcoma.

The 5 cases of lympho- or reticulum-cell sarcoma occurred in 3 women and 2 men, whose ages ranged from 10 to 80 years. The preoperative course was mostly less than 6 months, only in one case being as long as three years. The symptoms were most often oppression dyspnea, cough, thoracic pains, fatigue, loss of weight, and sometimes swallowing difficulty. All tumors were located in the upper anterior region varying in size between an orange and a fist, and all were growing infiltratively. The tumor masses were soft, grayish white, sometimes nodulated, sometimes with necrosis and small cysts Histologically, only 1 of the cases was a pure lymphocytosarcoma, the others were reticulum-cell sarcomas of small, large celled or polymorphocellular type In 2 of the cases, the surgeon tried to remove the tumor radically, in 2, partial resection was performed The fifth patient was never operated upon The autopsy in this case did not reveal any dissemination of the tumor All of the patients operated upon, in spite of radiologic treatment, have died within two and one half years after the operation

The case of plasmacytosarcoma occurred in a man of 37, presenting a two month's history of pains in the left arm and shoulder The thoracotomy revealed a tumor, the size of a hen's egg, in the upper posterior mediastinum, surrounding the three uppermost thoracic vertebras, growing into the inter vertebral foramina and surrounding the intercostal nerves and the left brachial nerve plexus X ray exploration did not reveal any involvement of the thoracic vertebras, nor any signs of myeloma in other parts of the bone system No albumin was found in the urine Histologically, the tumor tissue was built up by pleomorphic, mitose containing but mostly well-recognizable plasma cells The tumor was considered inoperable and only a biopsy was taken The patient died of the tumor one and one half years after the operation

THYMOMAS

There is much controversy concerning the definition of the term thymoma Many authors have described malignant widely metastasing tumors, supposedly originating in the upper anterior mediastinum, as thymic carcinoma, and the term thymic lymphosarcoma has also been introduced Without a doubt, the majority of the so called thymic carcinomas have been either pulmonary carcinomas with mediastinal extension or malignant teratomas with one sided development of the epithelial component We, also, believe that upper mediastinal lymphosarcomas originate from lymph nodes in this region and not from the thymic tissue Thus, we entirely agree with the definition recently given by Seybold and associates,[4] " thymoma is a slowly growing tumor of the thymus which has arisen from both the epithelial and lymphocytic elements of the thymic parenchyma " A thymoma may grow infiltratively in adjacent structures and give implantation metastasis in the pleura or pericardium Clearly documentated cases of thymoma, as previously defined, with extrathoracic metastasis, are—if existent—extremely rare

It has been suggested that those thymomas which are associated with myasthenia gravis fall into a distinct group In all cases of myasthenia gravis, the tumors seem to have fulfilled the criteria given previously, and no case with extrathoracic metastasis is clearly documented However, most thymomas with these morphologic and biologic properties are not associated with myasthenia gravis, and in our opinion it is impossible to find any histologic difference between the two groups

Thymoma constitutes a rather large proportion of the mediastinal tumors and cysts In the previous series, presented in Table II, it ranks fifth, in our series cases of thymoma come third after those of neurogenic tumors

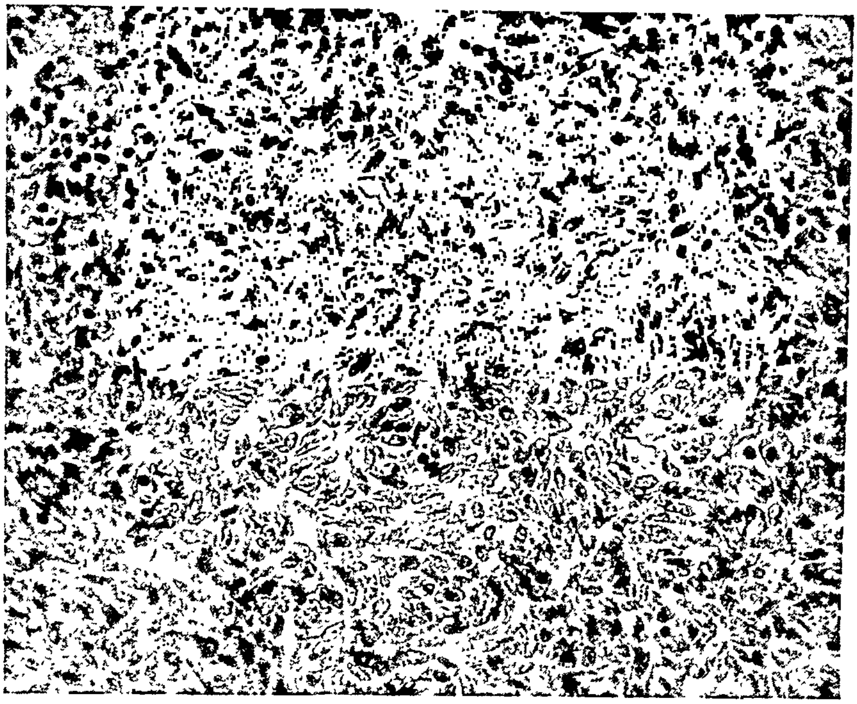


Fig. 10.—Thymoma of epithelial type. Note slight admixture of lymphocytes in the upper left corner. (van Gieson stain, ×280.)

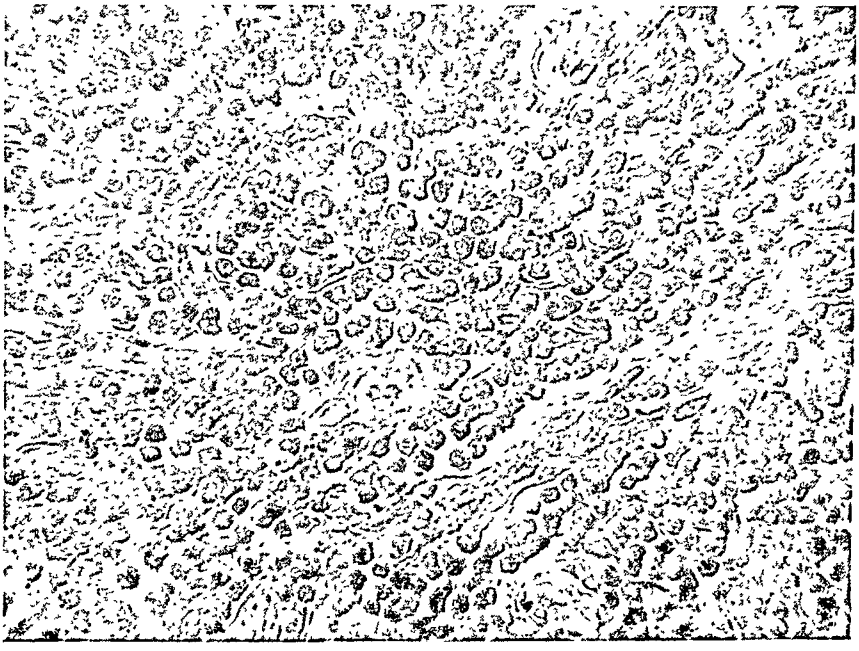


Fig. 11.—Thymoma of lymphocytic type with a perithelial arrangement of the thymo-epithelial cells (so-called reticular perithelioma of the thymus according to Pope and Osgood). (van Gieson stain, ×560.)

and bronchial cysts, which makes it more common than teratomas. Because of the confusion as to the definition of thymoma, it is impossible to give even an approximate evaluation of the number of cases of thymoma described or referred to in the literature.

This series includes 19 cases of thymoma. The sex distribution is even (9 men and 10 women). The ages range between 20 and 67 years. The mean age for both sexes is 47 years, but is 42 for men and 52 for women.

Thymomas are predominantly located in the upper anterior mediastinum, but location in the pulmonary hilar region is not rare, and even thymomas located on the neck and in the anterior inferior mediastinum have been described in the literature (Alter and Onato,[1] Ringertz,[41] Hardymoon,[21] and others). In this series, the site was the thymic region in 16 cases and the pulmonary hilar region in 3 cases.

The size varied between that of a mandarin orange and a large grapefruit (12 by 10 by 8 cm.). The tumors were rounded or flattened, often shaped as the thymus. The gross picture was characteristic in all cases: a nodulated tumor with broad streaks of fibrous stroma surrounding islands of soft grayish white tumor tissue. The latter may show cystic degeneration (in about one third of our cases) and cholesterol and lime salts may be deposited in the stroma. Infiltrative growth into adjacent structures was reported by the surgeon in only 1 case, and we were not able to establish this phenomenon, microscopically, in any other case. In this aspect, our series differs from that of Seybold and co-workers,[48] who found infiltrative growth in one fourth of their cases. In the previously mentioned case of invasive tumor, there were also pericardial implantation metastases, and in one other case, multiple pleural implants.

Microscopically, all the tumors in our series presented principally the same picture, the growth being made up of lymphocytes and thymo-epithelial cells. However, the proportion between the two kinds of cells varied very much, not only in the different tumors, but also in different parts of the same neoplasm. In 5 cases, the lymphocytes were definitely dominating, 5 showed about equal distribution, and 9 were predominantly epithelial. In the latter group, areas of purely epithelial tissue occurred (Fig. 10), but we always found other parts with lymphocytic admixture. In the epithelial areas, the fusiform cells showed a tendency to form knots and whorls, often with a pericapillar arrangement. We also found this whorl-like pericapillar proliferation of thymo-epithelial cells in an otherwise very lymphocyte-rich thymoma (Fig. 11—This picture probably corresponds to Case 6 in Pope and Osgood's[39] series of "reticular perithelioma" of the thymus). Hassall's corpuscles, more or less well developed, were found in 5 (nearly 25 per cent) of our cases, which tallies well with the frequency in the series of Seybold and associates.[48] A malignant cell picture was present in 2 cases which were not identical with those showing invasive growth and metastases. Thus, some sign of malignancy was found in 4 out of 19 cases.

The preoperative course was, in 5 of our cases, domiated by more or less pronounced symptoms of myasthenia gravis, in 1 case for twelve years. In 3 cases without this disease, there were only slight local symptoms such as pains and dyspnea. In 1 case, the thymoma was accidentally discovered during the course of agranulocytosis. In the remaining 10 cases, the patients were asymptomatic and the tumor was discovered by mass radiography.

Thus, we have 5 cases with preoperative myasthenia gravis. In another case, this disease developed after the operation and was obviously associated with the outgrowth of pleural metastases. These were removed, but the patient died four years after the first operation from myasthenia gravis. The autopsy revealed a local recurrence also. This makes, in all, 6 myasthenia gravis cases among 19 cases of thymoma; i.e., 32 per cent, a much lower figure than in the series of Seybold and co-workers[18] (75 per cent). The frequency of thymoma in myasthenia gravis is at least 15 per cent (Good[21]), in a Swedish series (Ringertz[41]) the percentage was 33. Four of the 6 patients with myasthenia gravis in our material were women with an average age of 49 while the two men were aged 20 and 21.

In all of our 19 cases, a mediastinal tumor was recognized roentgenologically.

Two of the patients were never operated upon, in 1 case because the woman died of myasthenia gravis before the planned operation could be performed, and in 1 case, the patient died of agranulocytosis. In 1 of the 17 operated cases, a biopsy was performed without further surgery (due to an erroneous first diagnosis of lymphosarcoma), and in 1 myasthenia gravis case with pericardial infiltration and metastases, a radical removal was impossible. In the remaining 15 cases, the tumor was radically extirpated.

The postoperative course in 11 of the 12 patients without myasthenia gravis, who were operated upon was without remark, and 10 of them are living free from symptoms one-half to four and one-half years after the operation. One patient has died of unknown cause and 1 by a traffic accident. Of the 5 patients operated upon with myasthenia gravis, 1 died postoperatively in respiratory insufficiency and 1 succumbed with myasthenia gravis due to metastases (see above). Of the remaining 3, 1 is quite symptom-free two and one-half years after the operation, 1 moderately improved after three years, and 1 slightly improved after one-half year.

THYMIC CYSTS

In the early literature, "thymic cysts" were frequently reported in syphilitic patients, especially in children with congenital syphilis. Nowadays, such lesions are rarely reported. On the other hand, a number of cysts claimed to have arisen from remnants of the thymic duct have been described in recent years. Krech and associates[31] (1954) collected 13 such cases from the literature and added 2 of their own. In a number of these collected cases, the cysts had a lining of high cylindrical, even ciliated epithelium, stratified or squamous epithelium. Many kinds of cysts may be located within the thymic region; teratomas of epidermoid cyst type, bronchial cysts, and cystic lymphangiomas. In our opinion, one must be very careful with the differential diagnosis. One cyst in this series we have classed as thymic cyst because of the widely distributed layer of thymic tissue with Hassall's corpuscles surrounding the thin fibrous, endothelium-lined wall. Due to the fact, however, that sparse bundles of smooth musculature were present in the wall, we suspect that this cyst might be an intrathymic lymphangioma. It occurred in a man of 61 years of age with two years' history of oppression and dyspnea. The cyst was of the size of a double fist, stalked from the thymic region and protruded into the right pleural cavity. The patient died five years postoperatively of gastric carcinoma. No autopsy was performed.

TERATOMAS

The mediastinum is, after the gonads, the most common site of teratoma in the human body. These neoplasms constitute a considerable proportion of the mediastinal tumors and cysts. Rusby,[43] in 1944, found 251 cases in the literature, and we have seen at least 100 more cases referred to after that year. The average percentage of teratomas in the series included in Table II is about 21, the teratomas ranking second after the neurogenic tumors. In our material, however, they rank fourth after the neurogenic group, bronchial cysts, and thymomas, with 10.3 per cent.

The frequency of malignancy among the mediastinal teratomas was 13 per cent in Rusby's collected material. Other literature compilations (Brewer and co-workers,[10] Laipply[32]) have shown 12.9 and 11.4 per cent, respectively. In some recent consecutive series, a higher percentage is shown, e.g., Hanner and associates[3] (1952), 33.3 per cent.

The sex distribution is even in the benign group, but it is pointed out that malignant teratomas predominantly occur in men (Fralic and Welsman[20], 1951, 8 males to 1 female). Most mediastinal teratomas occur in young adults. According to Willis,[60] the average age is 27 years. Laipply[32] states that 70 per cent of the patients are younger than 31 years and Rusby[43] that in 80 per cent of the patients the age was between 10 and 40 years.

The majority of recent authors consider the genesis of mediastinal teratoma to be associated with the development of the thymus (Schlumberger,[44] 1946). It is probable that all benign teratomas are tridermomas. However, in most cases, the ectodermal structures dominate so strongly that only a comprehensive histologic examination reveals the other structures. Because of this fact, and their common gross appearance of a cyst containing hairs and sebaceous matter, they have often been called dermoid cysts. The relatively high frequency of endodermal structures and, above all, of pancreatic tissue in the mediastinal teratomas has been pointed out in the literature. It is also stated that the malignant teratomas in this region lack ectodermal structures and that their epithelial component is predominantly adenocarcinomatous. The mesodermal structures are mainly low differentiated, though occasionally islets of cartilage and cysts of endodermal type may occur. Rare cases with a predominance of choriocarcinomatous tissue have been described in males (Laipply and Shipley,[33] 1945).

Our series contains 14 benign and 2 malignant teratomas, which gives a malignancy percentage of 12.5, which agrees with the figures of Rusby,[13] Brewer and Dolley,[10] and Laipply[32].

The *benign teratomas* showed an even sex distribution (7 males, 7 females), and an average age of 30. The location was the thymic region in 10 cases, the pulmonary hilus region in 3, while the site in 1 case was the costovertebral sulcus at the level of the third to the seventh thoracic vertebra. (This posterior location is rare, Rusby[13] reports only 3 such cases out of 251.) The size varied between duck's egg and melon, one-half of the specimens being at least the size of a grapefruit. All the benign tumors were well delineated and all but 1 had the gross appearance of a cyst with more or less pronounced mural nodule or nodules. The content was hairs and sebaceous matter in 10 cases, detritus only in 3. In no case were any teeth observed. One tumor in the thymic region was polycystic with solid tissue between the cysts. In 1 case, there was a fistula between the cyst and the main bronchus; this patient had expectorated hairs and showed multiple pulmonary abscesses.

Microscopically, 11 of these 14 tumors exhibited tissues from the three germinal layers in spite of the fact that the examination material was limited in several cases. In one well-examined case, only meso- and endodermal structures were found. As the endodermal structures in this cystic tumor did not appear as well-organized gastrointestinal mucosa, we have not classed the case as gastroenteric cyst. In 2 cases, endodermal structures were lacking, but in these cases the material was limited. The most common structures were epidermal with hair follicles, sweat and sebaceous glands; but cysts lined with respiratory epithelium, and with smooth musculature and mucous glands in the wall, were just as common. Cysts lined with epithelium of enteric type occurred in 7 cases, well-developed pancreas tissue in 3. Glia tissue was found only in 5 cases (36 per cent). Willis[55] states that its frequency in all teratomas is at least 80 per cent. We were not able definitely to identify nerve cells in any case. Cartilage and bone were present in 6 cases. Striated musculature and pigmented epithelium were found, each in 1 case.

Normal thymus tissue was found adjacent to the capsule in 6 of the cases, including 2 with pulmonary hilar position. No doubt this phenomenon would have been noted more frequently if we had been able to examine all the tumors in toto.

In 4 cases there were no symptoms, the remaining patients had a history of thoracic pains, dyspnea, and cough. In the bronchial fistula case, the patient had recurrent pneumonia and had expectorated hairs. Most of these tumors are obviously slow growing, 5 tumors—less than the size of a fist at operation—had been discovered three to eight years previously.

All the tumors were radically removed. One patient could not be traced afterward. All the others are living free from symptoms, 5 months to 10 years after the operation.

Our 2 cases of *malignant teratoma* occurred in men, aged 30 and 36, respectively. In both cases, the tumor was located in the thymic region, being about grapefruit sized, and showing a pronounced infiltrative growth In 1 case, where the neoplasm could be nonradically removed, its tissue was predominantly solid, grayish white and soft in consistency

Microscopically, 1 of the cases presented an entirely dominating epithelial component of highly malignant aspect, growing in festoons and *adenomatous* structures The sparse connective tissue was loose, myxomalike, without any cartilage or bone differentiation However, 1 small cyst with highly differentiated squamous epithelium was found The other tumor also presented, besides a low differentiated malignant epithelial proliferation, more differentiated *endo- and mesodermal structures, such as cysts with* ciliated columnar epithelium, *smooth musculature, and* mature connective tissue

Both patients had a preoperative history of less than three months of pains, fever, dyspnea, and hemorrhagic cough In 1 case, the tumor was not radically removed, in the other, only a biopsy was performed Both patients died within six months after the operation

MEDIASTINAL "BRONCHIAL ADENOMAS"

We have in this series two specimens of mediastinal tumor which were morphologically and clinically benign Their histologic picture was, at least in most parts of the tissue, very similar to that of bronchial adenoma These tumors were well encapsulated, large as a cocoanut, and the surgeon did not find any connection between the tumors and the bronchial tree We have not been able to find any identical cases described in the literature

Our material contains 1 more case of epithelial tumor, definitely primary, in the upper anterior mediastinum, but with a clearly malignant histologic picture The structure was, in this case, in principle similar to that in certain parts of one of the benign tumors mentioned before On the other hand, the epithelial component, growing in festoons and alveolar structures, presented a picture very similar to this component of a malignant teratoma As we were unable to establish organoid features in any part of this tumor, we have not classed it as a malignant teratoma but prefer to look upon it as a histologically malignant variant of the first mentioned tumors

We give here a short review of the findings in these 3 cases

CASE REPORTS

CASE 1 —*A man, aged 64 had a six months' preoperative history of* dyspnea and severe cough Roentgenologically, a large upper anterior mediastinal tumor was shown The thoracotomy revealed a well encapsulated growth, large as a large grapefruit, adherent to the right lung, dislocating the heart and the big vessels to the left The tissue was soft, solid, yellowish white with some necrotic patches The tumor was radically removed and the patient is free from symptoms five and one half years after operation *Histology* In some parts the tumor tissue was built up by monomorphous polyhedral epithelial cells with small round nuclei The cells were arranged in rounded clusters encased by narrow streaks of vascular stroma This picture was quite similar to that of bronchial adenoma of the carcinoid type (Fig 12, *A*) *In other parts, the* epithelial clusters merge into extensive solid masses penetrated by stroma streaks (Fig 12, *B*) No mitoses or any infiltrative growth in the capsular region were found

CASE 2 —A woman, aged 43, had no preoperative symptoms The tumor was discovered by mass radiography six months before operation The tumor was located in the posterior mediastinum and bulged into the left pleural cavity behind the heart. It was well encapsulated with a coarsly nodulated surface. The interior tissue showed cystic degeneration leaving papillary nodules of tumor tissue next to the capsule. The growth was radically removed and the patient was free from symptoms six years after operation. *Histology:* In some areas, this tumor presented the same picture as in the first case (see Fig. 12, *B,* and Fig. 13, *A*) but in other parts there was a peculiar structure with elongated cells showing a palisaded or rosette like nuclear arrangement (Fig. 13, *B*).

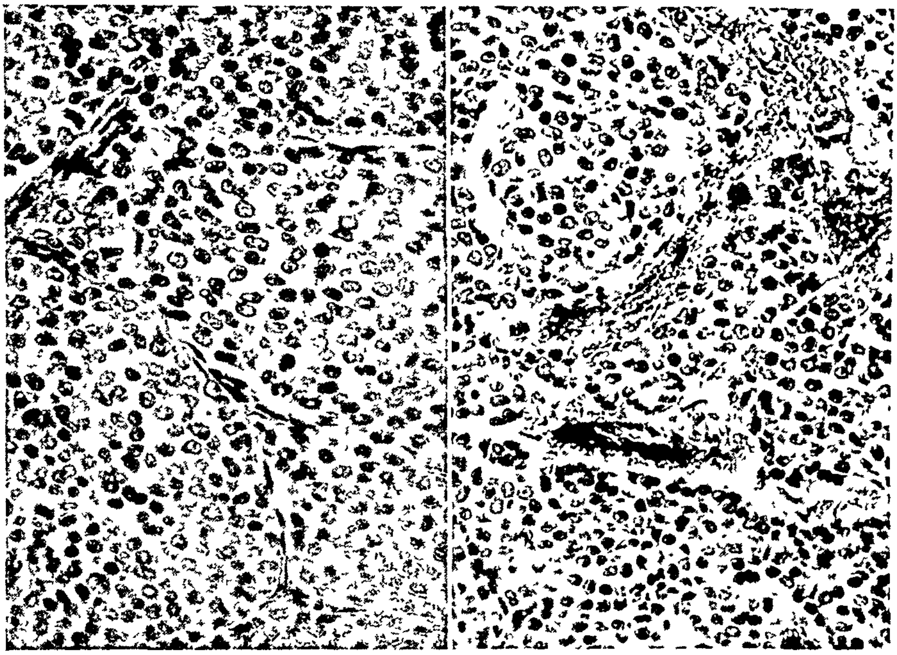
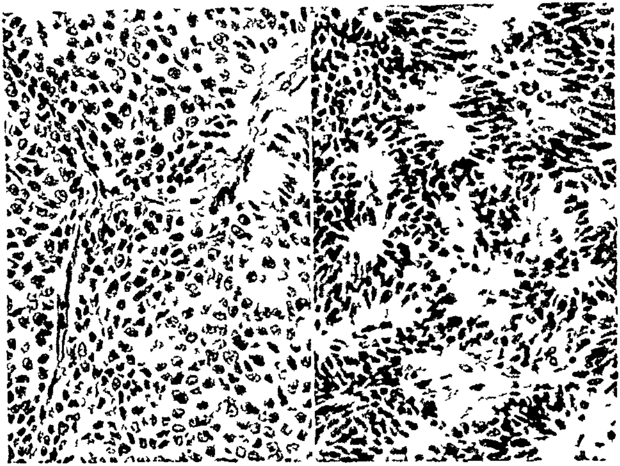


Fig. 12.—Case 1. Bronchial adenoma of the mediastinum. *A,* Solid clusters of tumor cells (carcinoid type); *B,* another part of the same tumor: diffuse cellular arrangement and sparse narrow stroma streaks. (van Gieson stain, ×280.)

Case 3.—A man, aged 39, after six months' symptoms of progressive dyspnea and arm edema, had a large upper anterior mediastinal tumor discovered by x-ray. A puncture biopsy showed a malignant, predominantly epithelial tumor, possibly of the teratoma type. The patient was treated radiologically for two years and ten months. X-ray revealed a slow progression of the growth, and due to the progressive dyspnea, it was decided to operate. At thoracotomy, more than three years after the first symptoms, an infiltratively growing retrosternal tumor was found. It was as large as a cocoanut, and bulged into the right pleural cavity and invaded around the big vessels and into the pulmonary hilar region. The patient died postoperatively and the autopsy showed remaining tumor tissue in the hilar region but no metastasis. *Histology:* The solid tumor had a uniform structure throughout. It was built up by epithelial cells growing in festoons and alveolar formations in a scanty loose nonmyxomatous stroma. There was an evident cellular polymorphism and many mitoses (Fig. 14). The arrangement of the epithelial cells was rather similar to that in Case 2 (see Fig. 13, *B*).

The pathogenesis of these tumors is difficult to interpret. They cannot possibly be exclusively centripetally growing bronchial adenomas, as none of them showed any anatomic connection with the bronchial tree. In Case 2, the gross aspect of the tumor might suggest the formation of an adenoma



A B

Fig 13 —Case 2 Bronchial adenoma of the mediastinum *A* Same type of tumor tissue as in Fig 12 *B* *B*, Another part of the same tumor with a rhythmic nuclear arrangement and formation of rosettes (van Gieson stain ×280)

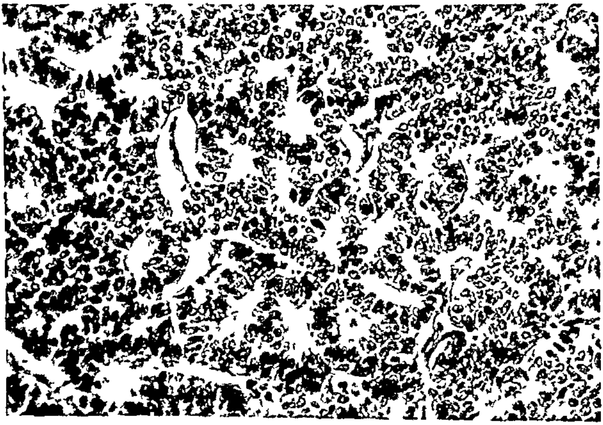


Fig 14 —Malignant mediastinal epithelioma probably belonging to the bronchial adenoma group Note a certain similarity in the cellular arrangement to that in Fig 13, *B* (van Gieson stain, ×280)

in a bronchial cyst, but histologic examination did not reveal any rest of bronchial cyst wall in the periphery. Possibly these tumors are teratomas with a one-sided differentiation, in which only a tissue, derived from the embryonal bronchial anlage, has been developed. Such an interpretation would explain the fact that the picture in Case 3 is so very similar to the epithelial component in malignant mediastinal teratoma.

BRONCHIAL CYSTS

These cysts are rather common mediastinal growths. Dodds[17] collected 85 cases in the literature up to 1950, and after that time at least 10 cases have been published. In the combined series recorded in Table II, they rank third after neurogenic tumors and teratomas. They occur characteristically in the posterior mediastinum at the level of the tracheal bifurcation and pulmonary hilum. On the basis of their location, Maier[37] has classified them into four groups; paratracheal, carinal, hilar, and paraesophageal.

The origin of bronchial cysts has been attributed to displacement of embryonal tissue. They are thought to arise from an additional budding of the foregut, equivalent to the respiratory anlage, which is carried down into the mediastinum. Thus, their development should relate them to accessory lobes of the lung.

Only a few of the cases reported in the literature have shown bronchial communication (e.g., one of Allison's[3] cases). Laipply[32] is of the opinion that such cases are bronchial diverticula and not real malformation cysts.

In our series, we have classed 21 specimens in this group. Nineteen of them presented the classical picture of bronchial cyst whereas 2 were multicystic formations with a structure resembling a malformed embryonal lung. We have not been able to find any cases in the literature similar to this latter group.

The 19 first mentioned cases occurred in 12 men and 7 women, whose ages ranged between 16 and 52, with an average age of 37 years. All but 4 were located in the posterior mediastinum. Eight cysts were located above, 5 below the level of the pulmonary hilum, and 6 in this level. As many of the cysts were fairly large, we found it difficult to group all of our cysts according to Maier's system. However, one was definitely paratracheal, three carinal, and six hilar. Their size varied between that of a hen's egg and a melon, 8 being at least grapefruit sized. Grossly, they were unilocular, thin-walled, the content being either clear, mucinous or brownish, or detrituslike (often mistaken by the surgeons for dermoid cysts). In 1 case, the cyst presented a narrow communication with the bronchial tree. We do not consider this goose egg sized cyst to be a bronchial diverticulum, especially as irregular patches of cartilage occurred in its wall.

The microscopic picture is characteristic, showing a lining of well-developed respiratory epithelium, often, in small areas, showing metaplasia toward squamous type. The thin fibrous wall always contains streaks of smooth musculature and mostly mucous glands. In 5 of our cases, irregular patches of adult cartilage were found in the wall. In 3 cases, we found small accessory lumina with respiratory epithelium included in the wall.

In 8 of our 19 cases (42 per cent), the cyst had not caused any symptoms and had been discovered by mass radiography (more than five years before the operation in 6 of these 8 cases). This percentage tallies well with Hirschfeld's statement that 60 per cent of the bronchial cysts give symptoms. In the other 11 cases, there was a preoperative course of up to six years with slight troubles, mostly cough, dyspnea, and oppression. All the patients had radical operations performed and are alive and well, ten months to ten years after the operation.

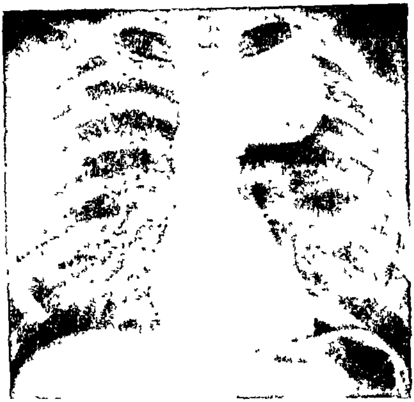
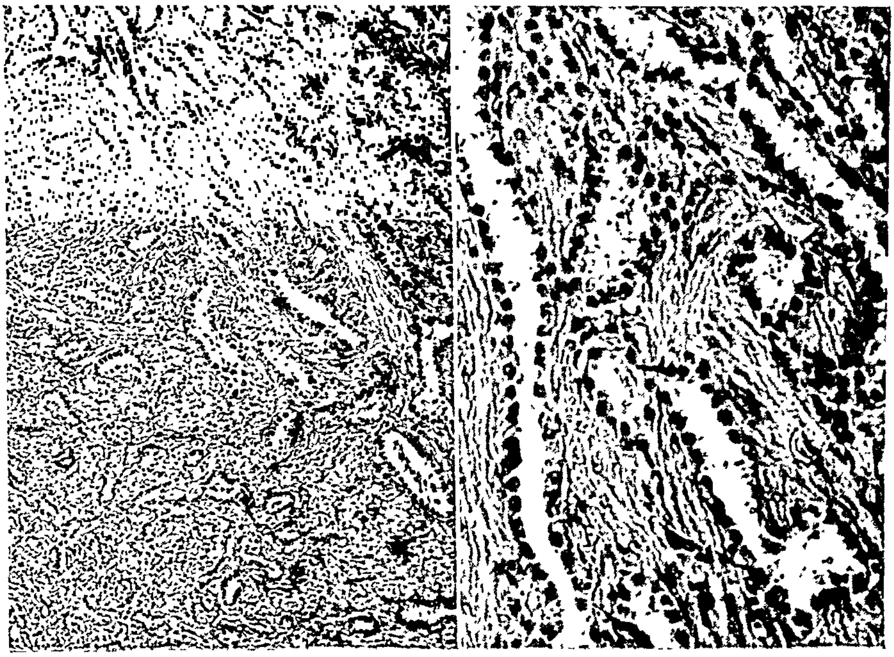


Fig 15—Roentgenogram of a malformation tumor of the "lung anlage" type. (See Fig 17)

The 2 multicystic specimens, mentioned previously, are very interesting, because they confirm the occurrence in this region of cysts with a structure resembling that of an embryonal lung. This is in consonance with the theory that bronchial cysts develop from an accessory pulmonary anlage. They occurred in one man and one woman, aged 20 and 32, respectively. They had both the same location, superiorly in the costovertebral sulcus, one plum and one grapefruit sized (Fig. 15). They were separated from the lung and showed no communication with the bronchial tree. Grossly, these formations were well *encapsulated, the cut surface having a honeycombed appearance with multiple,* small cysts. Microscopically, the 2 specimens seemed to reproduce different stages of the embryonal pulmonary development. Thus, in the small one the stroma tissue was built up by fibrous tissue and smooth musculature (Fig. 16). Encased in this tissue were multiple, small, elongated, and often bifurcated lumina lined with rather large, cuboidal, or low cylindrical nonciliated cells, resembling the cells in the early embryonal bronchial buds. No mucous glands or cartilage was present. The other specimen represented a more mature structure (Fig. 17). Here the cystic spaces were larger and covered by a mature respiratory epithelium. The fibrous stroma contained abundant streaks of smooth musculature often concentrically arranged around the cystic spaces. In this case, there was no admixture of cartilage or mucous glands, although the epithelium often contained mucous vacuoles.

In both these cases, the tumors grew slowly and without symptoms, and were discovered by mass radiography (in 1 of the cases, 9 years before the operation). They were easy to remove radically. The woman is alive and well five years after the operation. The man, however, (with the specimen



A. B.

Fig. 16.—Malformation tumor of immature "lung anlage" type. *A*, van Gieson stain, ×55. *B*, Detail of the upper left corner. (van Gieson stain, ×230.)

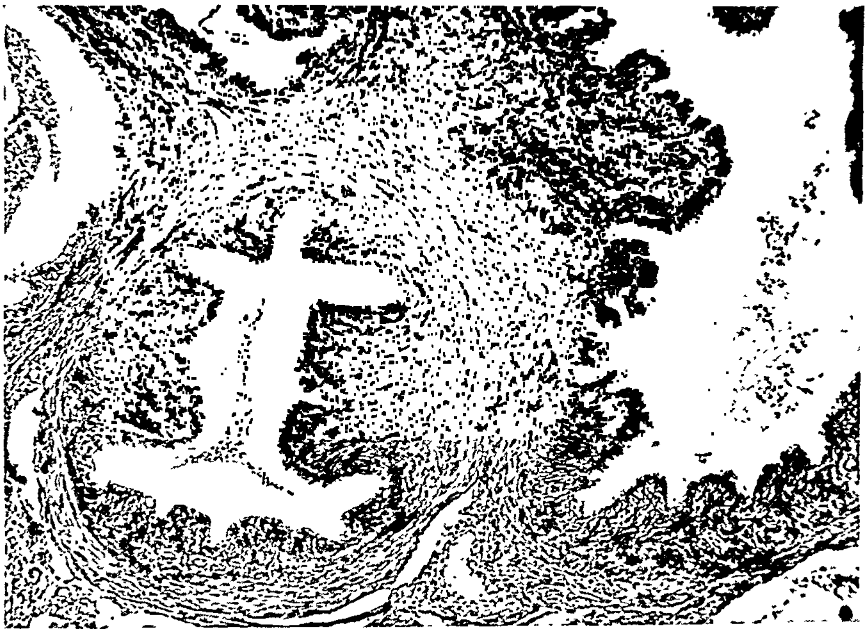


Fig. 17.—Malformation tumor of mature "lung anlage" type. Cystic spaces with cylindrical epithelium and concentrically arranged streaks of smooth musculature. (van Gieson stain, ×55; same magnification as in Fig. 16, *A*.)

of more adult type) developed a rapidly growing testicular tumor five months after the operation which was extirpated and showed the typical picture of seminoma. He died with signs of metastases seven months later. Unfortunately, no autopsy was performed. We feel strongly that the mediastinal affection with its definitively benign structure was in no way related to *the malignant* testicular tumor.

PERICARDIAL CYSTS

A fairly large number of thin-walled cysts with endothelial lining and a location close to the pericardium have been classed under this heading in the literature. Thus, Hirschfeld,[26] in 1951, was able to collect 72 such cases. Occasionally, these cysts show communication with the pericardial sac. In such cases, it is almost impossible to differentiate between pericardial diverticulum and real malformation cyst, due to deficient fusing of the primitive celomic lacunae. Moreover, some authors (e.g., Lambert,[34] and Leahy and Calver[35]) consider pericardial cysts and diverticula to be the same thing.

It may also be difficult to differentiate between pericardial cyst and lymphagioma. Grossly, these two growths may be very similar. However, in our opinion, it is possible to separate them microscopically, because the lymphangiomas nearly always have streaks of smooth musculature and small lymphocyte infiltrates in the walls. We also feel that the lining cells in *pericardial cysts, at least in patches, are cuboidal rather than flat endothelial.* In the combined materials, presented in Table II there are 1.6 per cent lymphangiomas and 10.4 per cent pericardial cysts, whereas in our series the figures are 4.5 per cent and 3.9 per cent, respectively. We suspect that some of the pericardial cysts frequently reported in the literature really are lymphangiomas.

Our series contains 6 cases, 4 men and 2 women with ages ranging between 31 and 56, in average 41 years (Hirschfeld[26] found no case in the literature younger than 27). The cysts were all located in the anterior inferior mediastinum, in 4 cases on the right and in 2 on the left side of the pericardial sac. The largest cyst, right-sided, measured about 24 by 20 by 20 cm., the smallest one was the size of a hen's egg. In one case, the cyst showed a 1 cm. wide pericardial communication. In another case, there was a small cyst which stalked from the main one. All the cysts contained a clear nonviscous fluid and had a thin, purely fibrous wall with a lining of single row of cuboidal or flat cells.

In 5 of our 6 cases, there were no preoperative symptoms, the cysts being discovered by mass radiography (Hirschfeld[26] reports a symptom-free preoperative course in 39 of his 72 literature cases). Our remaining case had a six months' history of severe cough and slight pains. All the cysts were removed radically and all the patients were alive and well, three months to seven years after the operation.

UNDEFINED CYSTS

Our series contains 4 cases with cystic affections in the upper anterior mediastinum, where we have not been able to classify the specimens definitely, in spite of the fact that the histologic material was rather extensive. One of these specimens, a grapefruit-sized multicystic formation removed from a man aged 58, could possibly be a so-called "burned out thymoma" (Blalock[8]). This patient had no signs of myasthenia gravis at admission or in the history. In 2 other cases, the cysts, about the size of a hen's egg, had a thick fibrous wall with cholesterin granulomas but without any epidermal or other organoid structures. However, we cannot exclude the possibility that they are adult teratomas of the "dermoid cyst" type with strong regressive changes. The last specimen, large as a mandarin orange, had a thin, fibrous wall with rather abundant lymphocyte infiltrates but lacked entirely epithelial or endothelial lining.

SUMMARY

This is a survey of 155 patho-anatomically verified primary mediastinal tumors and cysts, treated at the Clinic of Thoracic Surgery at Sabbatsberg Hospital during the period 1944 to 1954. Table I gives the composition of the material, and on page 458 the mediastinal affections, excluded from or missing in the series, are recorded. A comparison between the composition of this series and those of some previous investigators is given in Table II.

Not less than 61 per cent of these tumors and cysts were discovered by mass radiography, and Fig. 1 gives the relation between the frequency of mass radiography in Sweden and the number of growths in this series, thus discovered.

Location, gross anathomy, histology, age and sex distribution, clinical course, and the result of follow-up are recorded.

This series includes a number of rare tumors, some being difficult to classify and the investigation has in certain details given results, partly new and partly disagreeing with previous experience. Thus, all the 3 cases of ganglioneuroblastoma ran a benign course, which does not tally with the findings of, e.g., Ackerman and Taylor.[1] Among the hemangiomas (recorded on page 468) there is 1 case of hemangiopericytoma and another peculiar melanin-pigmented specimen, possibly a neurovascular malformation. The series includes 1 case of plasmacytosarcoma with no signs of multiple myeloma.

All tumors classed as thymomas have been diagnosed in accordance with the restrictive definition given, among others, by Seybold and associates,[48] and in no case were there signs of extrathoracic metastasis. Only 32 per cent of the thymoma patients had myasthenia gravis (Seybold and co-workers[48]; 75 per cent).

The material includes 2 tumors, quite unconnected with the lung or bronchial tree but with a structure very similar to that of bronchial adenoma (Figs. 12 and 13). In another similarly located, infiltratively growing tumor the microscopic picture was a malignant counterpart of the former (Fig. 14). The possibility is emphasized that these tumors are teratomas with a one-sided development, arising from the bronchial anlage. We have not been able to find any identical case in the literature.

Besides 19 cases of classical bronchial cyst, the series includes 2 multicystic specimens located in the costovertebral angle and with a structure similar to a lung anlage (Figs 16 and 17) The occurrence of such mediastinal malformations is in consonance with the theory that bronchial cysts develop from a pulmonary anlage We have not seen any identical cases in the literature

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FIBROUS DYSPLASIA OF BONE: ANALYSIS OF 15 CASES OF SURGICALLY VERIFIED COSTAL FIBROUS DYSPLASIA

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FIBROUS dysplasia of bone is characterized by the occurrence of one discrete skeletal defect, or of a few, or numerous such defects. Brown-to-yellow patches of cutaneous pigmentation may accompany the bone lesions, especially in severe disseminated fibrous dysplasia. When, in addition to cutaneous pigmentation, patients having polyostotic fibrous dysplasia show signs of endocrine abnormalities, especially precocious puberty among girls, the condition is commonly referred to as "Albright's syndrome."[1] Excluding the 67 cases collected in the armed forces,[2] no sex predilection of fibrous dysplasia has been observed. The etiology of these bone defects is unknown; the weight of evidence indicates that they are due to a congenital anomaly in which the activity of the specific bone-forming mesenchyme is perverted.[3] The cause of the extraskeletal lesions is equally obscure. The disease usually appears early in life and progresses most actively during the patient's growth. The long-standing confusion of polyostotic fibrous dysplasia with the bone lesions of hyperparathyroidism should rarely be a problem when one considers the diffuse demineralization, chemical changes in blood, renal lithiasis, and the older average age of patients having hyperparathyroidism.

When a single bone only is involved, the lesion is called "monostotic," even though the defects occasionally are multiple. It is difficult to reach an accurate figure concerning the relative frequency of the monostotic as compared to polyostotic lesions. The literature is likely weighted in favor of the more dramatic full-blown examples of Albright's syndrome. Furthermore, total skeletal roentgenograms would be necessary to establish the authenticity of the lesions reported as monostotic. The consensus is that the solitary lesions are more common. Berger and Jaffe[4] have said that for each patient having fibrous dysplasia with full-blown Albright's syndrome there are 20 or 30 patients who have fibrous dysplasia with only one or a few bones involved.

The cutaneous pigmentation is owing to an increased deposition of melanin in the basal layer of the epithelium. These single or multiple brown-to-yellow discrete areas may be of any size. In the review of 256 cases by

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Pritchard,[5] pigmentation was recorded in 79 instances, although it was not mentioned in any of the 67 monostotic cases contributed by Schlumberger.[7] Pigmentation is much more likely to occur with severe polyostotic fibrous dysplasia, especially if endocrine abnormalities are present, than with the monostotic form, although it has been observed in association with the latter form.

Endocrine effects occur much more commonly among women than among men with polyostotic fibrous dysplasia. Precocious sexual development was observed in 37 women and 5 men, or 16.4 per cent of the series reviewed by Pritchard. Premature menstruation, early development of secondary sexual characteristics, and premature fusion of epiphyses resulting in adults of small stature are the common features. Rarely are retardation of puberty and hypogenitalism[6] exhibited in either sex. Hyperthyroidism seems to occur with extraordinary frequency among patients with polyostotic fibrous dysplasia, a total of 6 unequivocal examples of this association having been observed by 1951.[8]

A rather special type of fibrous dysplasia occurs in jawbones[4] and in bones at the base of the skull. The fibrous tissue of lesions in these regions, especially in the skull, tends to show much more metaplasia to bone than it does in other sites. This often produces roentgenologically dense, expanding lesions that are more or less heavily calcified. Many lesions of this type have been called "ossifying fibromas" and even "osteomas." The very appropriate term, "fibro-osseous dysplasia," has been suggested in these cases. Berger and Jaffe consider that in about 10 per cent of the cases of monostatic fibrous dysplasia, the lesions occur in bones of the head, and one half of the patients with polyostotic fibrous dysplasia will have one or more lesions in bones of the skull. Much evidence indicates, furthermore, that the condition included under the descriptive term "leontiasis ossea"[9] is due to fibrous dysplasia of bones of the skull and face. Some workers[10] also consider "cherubism" to be a manifestation of fibrous dysplasia.

Any bone may be affected in fibrous dysplasia. Several authors have stressed the occasional tendency for the polyostotic lesions to be confined to one side of the skeleton. In the collected series of all types of fibrous dysplasia reviewed by Pritchard, he tabulated the distribution of lesions as follows: lower limbs, 118 cases; upper limbs, 87; skull, 52; face, 31; and trunk bones, 49. Schlumberger noted that a rib was the site of involvement in 29 of 67 isolated lesions diagnosed as fibrous dysplasia of bone. More recently Hochberg[11] has found that, including Schlumberger's group of 29 cases of fibrous dysplasia, the total of 35 cases comprised one third of all cases of benign costal tumors which he found reported in the literature.

The relatively high frequency of costal involvement in both monostotic and polyostotic fibrous dysplasia makes this lesion of significance to the general surgeon. The lesion is often asymptomatic and discovered on routine roentgenograms of the thorax, whereupon a decision regarding therapy must be made. For these reasons a study of our surgically verified costal lesions of this type was undertaken.

PRESENT SERIES

A review of the series of surgically treated costal tumors at the Mayo Clinic showed 15 examples of fibrous dysplasia. Thirteen of these lesions were excised intact along with the involved segment of rib, and curetted fragments were obtained in the other 2. The markedly selected nature of these cases limits their value in understanding fibrous dysplasia as a whole. In most cases of polyostotic fibrous dysplasia, costal lesions have not been treated surgically. For example, in the report on Albright's syndrome from the Clinic by Dockerty and associates,[12] 4 of 6 patients whose cases were presented had costal lesions which were not subjected to operation. The surgically excised costal lesions afford a unique opportunity, however, to study intact gross specimens, since the average fibrous dysplasia of other bones is usually excised piecemeal with a curette if removed at all.

Age and Sex.—The ages of the 15 patients varied from 18 to 65 years, and 12 of them were more than 30 years of age. The relatively advanced age of this group is partially explained by the fact that the lesions were accidental roentgenographic discoveries in 7 instances. Several of the other patients had had symptoms for a long period. Eight patients were women and 7 were men.

Clinical Features.—Three patients had noted some thoracic discomfort for periods of ten to fifty years; 5 had had thoracic pain or soreness for periods ranging from one week to four years, and 7 had not had any symptoms. Pathologic fracture had occurred in only 1 case.

Physical examination was noncontributory in 10 cases, although in 1, the patient had had multiple subcutaneous lipomas over the surface of the thorax and abdomen. Three patients had palpable enlargements or irregularities of the affected ribs and local tenderness was present in 2. Patches of cutaneous pigmentation were not recorded in any case of this series.

Roentgenographic Findings.—A frequent manifestation of fibrous dysplasia of the ribs consists of moderate expansion of a long segment of the rib with the affected bone having a uniform ground-glass appearance so that the cortex and spongiosa cannot be distinguished. This type of fibrous dysplasia does not have a cystic appearance and often more than one rib is affected. The correct diagnosis is usually quite obvious and the patients seldom undergo operation for the condition. For that reason no patients having lesions of this type are included in the group that underwent operation.

The roentgenograms revealed the bony defect in each case. The characteristic lesion produced expansion of the rib with an irregularly attenuated, though intact, cortex. The expansion was usually toward the thoracic cavity so that even when the lesions were large the tumor often was not evident externally. The expanded portion was basically radiolucent but frequently appeared trabeculated and showed a fuzzy, patchy type of density. The ends of the affected zones were sharply demarcated from the uninvolved portions of the rib. The ribs with long zones of fibrous dysplasia became distorted in contour and expanded to variable degrees so that the normal parallelism to adjacent ribs was lost. In lesions with classic roentgenologic findings, the diagnosis of fibrous dysplasia can be made with considerable certainty. In

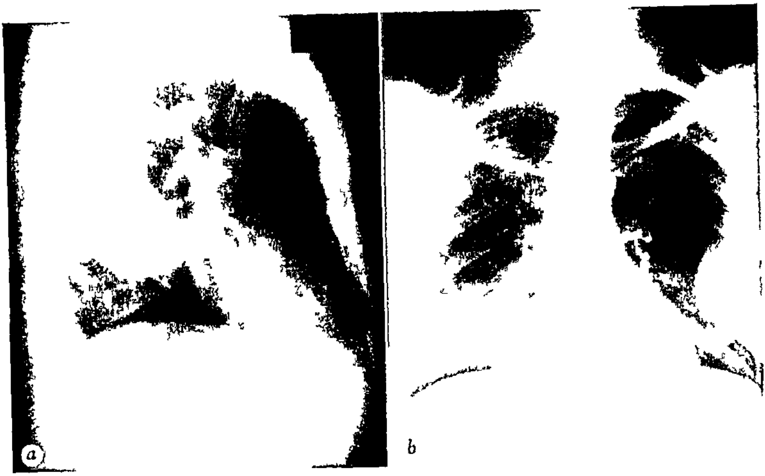


Fig 1, *a* and *b*—Fibrous dysplasia of the seventh left rib as seen in the posteroanterior and lateral roentgenograms

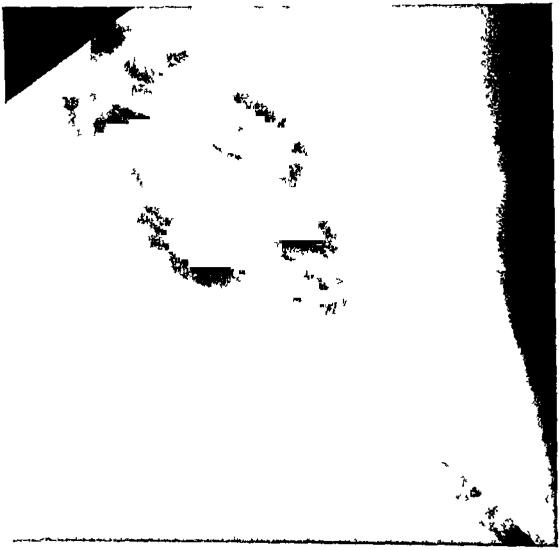


Fig 2—Fibrous dysplasia of long segment of left fifth rib

some cases, however, the roentgenologic defects may be mistaken for lesions such as central cartilaginous tumors, or myeloma, or eosinophilic granuloma (Figs. 1 and 2).

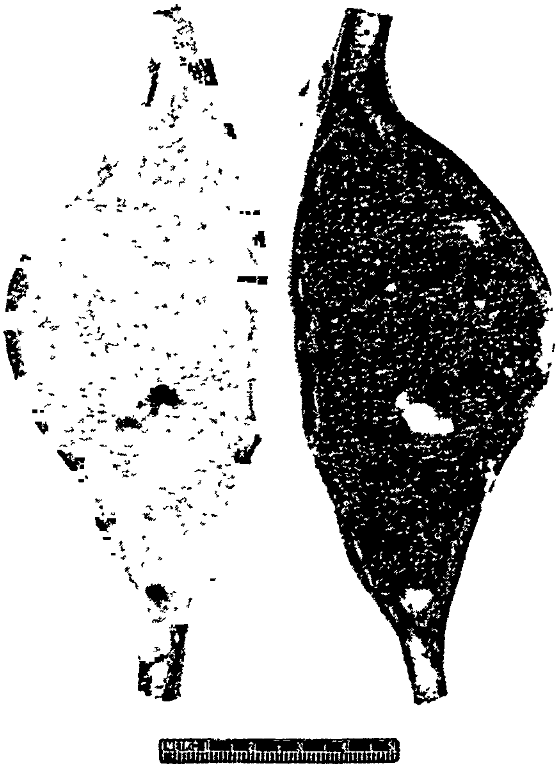


Fig. 3.—Surfaces produced by longitudinal section of the lesion shown in Fig. 1, *a* and *b*.

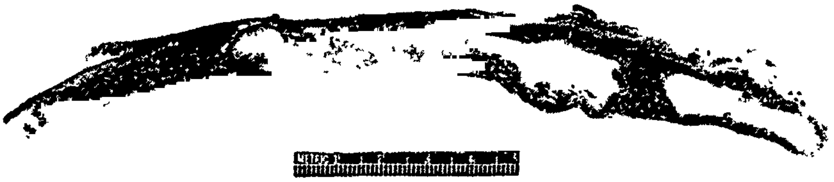
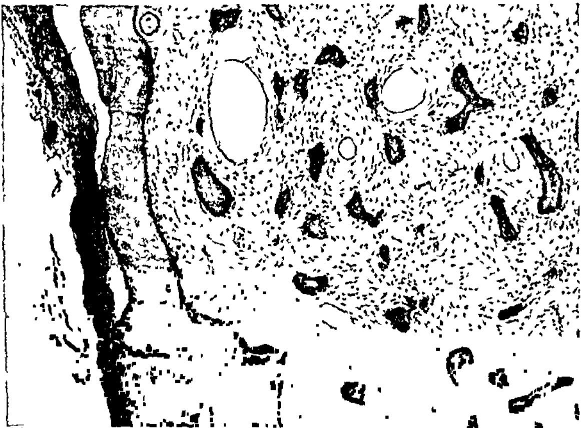


Fig. 4.—Only slightly expansile, partially cystic fibrous dysplasia of left fifth rib (same case as Fig. 2).

Fibrous dysplasia was apparently polyostotic in 3 of the 15 patients. One patient whose diagnosis was verified by biopsy had similar defects in two other ribs and a radiolucent area in the ilium. Another patient with expanding lesions of a rib and the transverse process of a lumbar vertebra was thought to have myeloma until the affected rib was curetted for biopsy. One patient with similar defects in two ribs had 1 of the lesions removed.

Pathologic Aspects.—The specimens removed (Figs. 3 and 4) varied in size from 5 by 1 by 1 cm. to 19 by 6 by 5 cm. They were uniformly central, expanding, fibrous masses. The expanded costal cortex showed irregular thinning, and its internal surface frequently exhibited corrugations, the shadows of which sometimes appeared as septa in the roentgenograms. Partially calcified osteoid masses in the fibrous tissue of 10 lesions imparted a gritty character to the cut surface. Cystic areas, which measured as much as several centi-

Fig. 5.



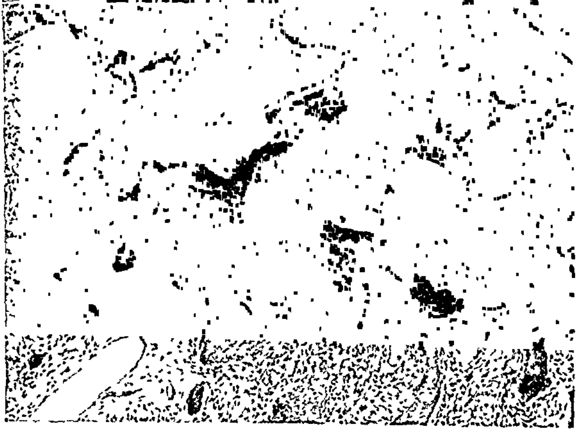


Fig 6.

Fig. 5.—Typical fibroblastic tissue containing irregular islands of new bone. The broad vertical band near one end represents the distended cortical shell. (Hematoxylin and eosin; ×75.)

Fig. 6.—More cellular area of fibrous dysplasia with larger *osteoid masses*. (Hematoxylin and eosin; ×75.)

meters in diameter in the larger lesions, were present in 6 cases. In 2 instances, the cysts comprised more than one-half the bulk of the area of dysplasia. Three specimens contained brown zones owing to deposits of hemosiderin. Although fusiform expansion was the usual gross configuration, 1 lesion involved 17 cm. of a rib but the rib had expanded to a maximum of only 1.5 cm.; another rib was distinctly dumbbell-shaped with two globular expansions connected by an isthmus of fibrous tissue. In all cases, the dysplastic tissue was distinctly demarcated from the adjacent normal bone.

The histologic features of fibrous dysplasia have been well documented. Briefly, the findings in this series (Figs. 5 and 6) were as follows: fibroblasts lying in a more or less dense collagenous matrix dominated the picture. The cellularity varied greatly from lesion to lesion and to a less extent in different portions of the same lesion. All lesions contained zones in which irregular osteoid masses had been laid down in the fibrous matrix. Slight to moderate calcification of these osteoid trabeculae usually had occurred. The amount of osteoid trabeculae varied considerably, and in several lesions central areas contained little or none. As pointed out by Schlumberger, these trabeculae which aid greatly in making the diagnosis tend to be most prominent in the peripheral zones. Cartilaginous zones were not found in any of the lesions. Focal collections of hemosiderin, usually within macrophages, were found in 7 lesions and, as already noted, they were prominent enough to produce grossly visible brown zones in 3. Benign multinucleated cells were present in 8 specimens and in several instances they were numerous enough in foci to simulate giant-cell tumor of bone superficially. Myxomatous changes were found in parts of the fibrous tissue of 7 of these costal tumors. This degenerative change apparently preceded cystic formation because it was especially prominent in the vicinity of gross and microscopic cysts. Inflammatory cells were rare; foci of lymphocytes and plasma cells were found in 1 lesion.

COMMENT

This series of cases of fibrous dysplasia of ribs emphasizes some of the features of this peculiar growth anomaly. Cystic formation may be a prominent pathologic characteristic of the gross specimen, and in rare instances, the dysplastic zone may be predominantly cystic. Although chondroid foci within the lesional tissue have been considered to give an important diagnostic clue in many cases of fibrous dysplasia,[13] their complete absence in this series is noteworthy.

The indolent nature of the pathologic process in this disease is suggested by the fact that no symptoms had been produced in 7 patients and several of the remaining 8 had had but slight difficulty for many years.

Once one is familiar with this entity the diagnosis is rarely difficult. The metaplastic osteoid trabeculae have a characteristic appearance and are not found in nonosteogenic fibroma. The lack of cellular anaplasia precludes the serious consideration of any malignant process. The collagen production in fibrous dysplasia contrasts with the lack of such production in diagnostic fields of a giant-cell tumor of bone, and this fact alone should differentiate the two conditions The lesions with large cystic areas may cause some confusion, but even in these the central expansile character of the process as observed roentgenologically and grossly, coupled with histologic evidence from the solid areas, ordinarily solves the problems

Regarding treatment of fibrous dysplasia of ribs, there may be some disagreement In examples of the polyostotic form of the disease, costal lesions rarely will require treatment With single or only a few lesions, the physician may not be absolutely certain of the diagnosis without pathologic study In such cases, a plea should be made for total excision of the involved regions if at all feasible Such surgical management would help to prevent contamination of the wound by malignant tissue if the lesion proves to be malignant The size of the lesions of several of our patients constituted adequate indication for removal Malignant change in fibrous dysplasia has been observed so rarely[14, 15] that this threat can scarcely be considered an indication for surgical intervention

SUMMARY

Fibrous dysplasia of bone is characterized by the occurrence of one, a few, or numerous discrete skeletal defects The lesions most commonly are limited to one bone in which case the condition is known as the "monostotic" type However, there are cases in which disseminated skeletal lesions have been associated with endocrine abnormalities and areas of pigmented skin The etiology of the bone defects is unknown, however, the weight of evidence indicates that the defects are caused by a congenital anomaly in which the activity of bone forming mesenchyme is perverted The extraskeletal lesions have an obscure etiology also It is a disease of early life and progresses most actively during the patient's growth Fibrous dysplasia may affect any bone The frequency of costal involvement is relatively high, which makes this lesion significant to the general surgeon The lesion is often asymptomatic, discovered on a routine roentgenogram of the thorax, and a decision regarding therapy must be made For these reasons a study of our surgically verified costal lesions was undertaken

Our study included 15 cases of fibrous dysplasia of ribs The ages of the patients ranged from 18 to 65 years Eight patients were women and 7 were men Clinical signs and symptoms were few Three patients complained of thoracic discomfort, 5 of thoracic pain or soreness of a mild nature, and 7 were free of symptoms Ten patients had no physical signs, 2 had local soreness, and 3 had noted some enlargement over the affected rib A roentgenogram of the thorax revealed the lesion in each case The lesions were found to be uniformly central, expanding fibrous masses, ranging in size from 5 by 1 by 1 cm to 19 by 6 by 5 cm The expanded cortex of the rib showed irregular thinning The internal surface of the cortex often exhibited corrugations, the shadows of which appeared as septa in the roentgenograms Partial calcification makes the lesion gritty and cystic areas were found in 6 cases In all cases the dysplastic tissue was distinctly demarcated from the adjacent normal bone. The main histologic feature is fibroblasts lying in a more or less dense collagenous matrix. The pattern is irregular, however, since the cellularity varies and regions of osteoid trabeculae and calcification may be found.

The diagnosis of this condition is not difficult if one is familiar with it but some disagreement exists regarding therapy. In examples of the polyostotic form, costal lesions rarely require treatment. If only a single lesion or a few lesions are present, pathologic study may be necessary before a positive diagnosis can be made. Therefore, a plea is made for total excision of the involved portion if at all possible.

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HEREDITARY HEMORRHAGIC TELANGIECTASIA

Report of a Case With Hemothorax

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MASSIVE hemothorax in a patient known to have hereditary hemorrhagic telangiectasia (Osler-Rendu Weber disease) has been previously reported only once We encountered this complication in a 44 year old man Its rarity and the difficult problems involved in its management have prompted this report

The first description of hereditary telangiectasia was written in 1865 when Babington[1] gave an account of recurrent epistaxis through 5 generations of a family The cases reported by Sir William Osler[2, 3] established the disease as a definite entity During the past fifty years, hundreds of case reports have appeared and more than 150 families have been described Bleeding from practically every organ has been reported in this disorder Other diseases have been described in association with hereditary telangiectasia but pulmonary arteriovenous fistula is the only associated disorder commonly seen More than one half of the pulmonary arteriovenous fistulas reported to date have been in association with hereditary telangiectasia

The name, hereditary hemorrhagic telangiectasia, first used by Hanes[4] in 1909, describes three of the criteria for diagnosis The triad of telangiectases of the skin, mucous membranes, or viscera, a tendency to bleed from these lesions, and a family history of intermittent hemorrhages is strong evidence for the disease If, in addition, the patient has a normal clotting mechanism, the diagnosis is established The chief obstacle to diagnosis is the failure of the physician to think of the disease These patients almost invariably complain of epistaxis which usually begins near puberty and recurs frequently thereafter Blood dyscrasias are easily ruled out by laboratory studies A careful search of skin and mucous membranes will reveal the lesions Bronchoscopy,[5] gastroscopy, or proctoscopy[6] may show telangiectases of the trachea, bronchi, stomach, or rectum

The lesions of hereditary telangiectasia may be located on the surface or in the parenchyma of any organ The visible telangiectases may be pin-point and flat but are usually several millimeters in diameter and slightly elevated Their color varies from cherry red to purple Those on the mucous membranes and skin usually appear near the age of puberty but have actually been noted

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at birth. Spider-like telangiectases are also frequently present. The absence of pulsation distinguishes these spider angiomas from those seen in liver disease.[7] Bleeding from viscera usually does not occur before the third decade of life. There is a tendency for new telangiectases to appear with increasing age. These nodular lesions blanch readily and may pulsate. Microscopic study shows vascular sinuses of irregular size and shape, lined by a thin layer of endothelium. Muscular and elastic coats are absent. Because of the thin walls and proximity to the surface, these sinuses bleed readily with slight trauma and, because of the lack of contractile elements in the wall, bleeding may not cease spontaneously.

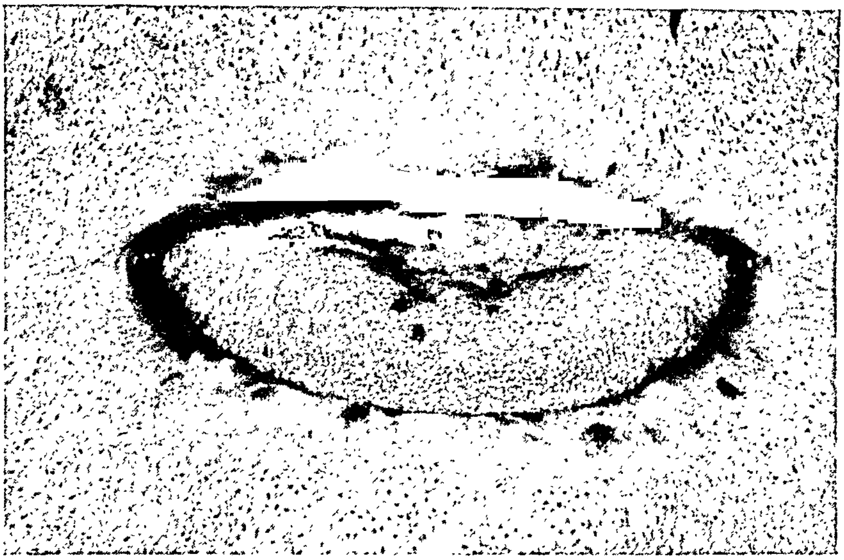

Fig. 1.—Typical lesions of hereditary hemorrhagic telangiectasia on lips and tongue.

Descriptions of the pulmonary arteriovenous fistulas found in this disease show them to be anatomically similar to the peripheral telangiectases. They are large sinuses interposed between arteries and veins. The afferent artery and efferent vein are usually dilated and thin walled. The danger of massive hemorrhage into the pulmonary parenchyma is ever present. In over one-half of the cases, the pulmonary arteriovenous fistulas are multiple. Goldman[8] believes that all congenital pulmonary arteriovenous fistulas are a localized manifestation of generalized hereditary telangiectasia.

Hereditary telangiectasia is transmitted as a simple dominant trait and affects both sexes. The children of a family in which one parent is affected, therefore, have a 50 per cent chance of inheriting the disease. Often the lesions are confined to the mucous membranes of the nose and mouth and epistaxis may be the only symptom (Fig. 1). In general, the earlier in life the telangiectases make their appearance the worse the prognosis.

The only known report of a child born to parents who were both afflicted with hereditary telangiectasia described the appearance of new telangiectases daily until death at less than 3 months of age.[9] The probability of such a lethal form of the disease in a child of parents who are both affected is one in four.

The predominant symptom of hereditary telangiectasia is recurrent painless hemorrhage. Epistaxis is such a consistent manifestation that one should question the diagnosis if the patient has no history of epistaxis. When bleeding is from a visible nodular lesion it may be seen to come in spurts. Bleeding may occur in the mouth, nose, stomach, bowel, kidney, bladder, liver, spleen, brain, or meninges. Some have noted an increase in size of the telangiectases and tendency to hemorrhage during menstrual periods.[10] Campbell[11] suggests that many cases of "essential hematuria" are really due to hemorrhagic telangiectasia. We suspect that the same is true of "spontaneous hemothorax." Massive painless gastrointestinal bleeding is very common.[12] The bleeding may lead to signs of anemia but this is frequently masked by the polycythemia which is an accompaniment of arteriovenous fistulas. Patients may, therefore, have a normal or high hemoglobin even after a hemorrhage.[13] Others alternate between polycythemia and a marked anemia.[14] Cyanosis, shortness of breath, clubbing of fingers, and a bruit are common in this group. Fatal hemoptysis from such a fistula has been reported.[15]

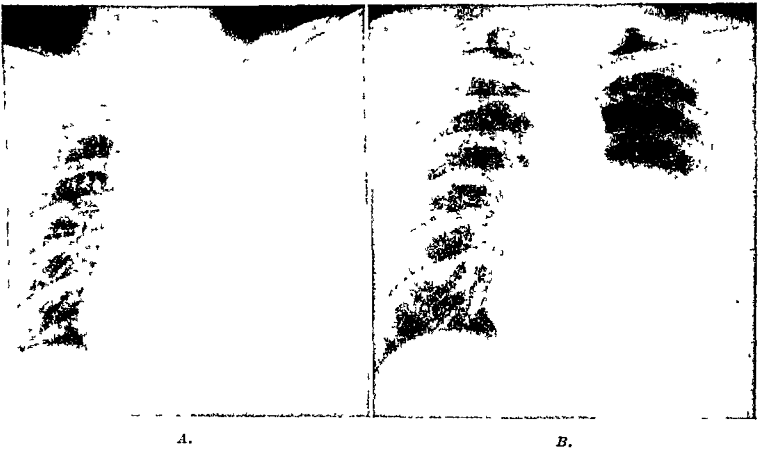
A. B.

Fig 2.—A, Roentgenogram on admission to another hospital showing opacity of left hemithorax of fluid density with slight displacement of the heart to the right. B, Roentgenogram on admission to Veterans Administration Hospital, McKinney, Texas, showing opacity of lower half of left chest with curved line typical of fluid

Treatment with rutin, irradiation, estrogens, ascorbic acid, moccasin venom, vitamin K, and other drugs has given poor results. Oral thrombin appeared to be effective in a case of massive bleeding from multiple telangiectases of the small bowel.[16] Because of the tendency for remissions and exacerbations in the hemorrhagic tendencies the evaluation of any therapy is difficult.

CASE REPORT

The patient was a 44-year old laborer, who had a sudden onset of shortness of breath and left chest pain on Feb. 5, 1952. He was admitted to a h[illegible]r with a hemoglobin of 9.8 Gm. and a thoracentesis yielded 250 c.c. of blood. Thoracentesis was performed the following day and 600 c.c. of blood removed. He was transferred to the Veterans Administration Hospital, McKinney, Texas on Feb. 13, 1952, with a diagnosis of "hemothorax, left, probably due to neoplasm of the pleura." Upon arrival the patient was acutely ill, pale, listless, with temperature 101.5° F., pulse 102, and blood pressure 105/70. There were signs of fluid in the left chest and the hemoglobin had dropped to 7 Gm. He had multiple small elevated cherry-red telangiectases on the tongue, buccal mucosa, lips, nasal mucosa, and skin. The skin telangiectases were most numerous over the face and trunk but there were some over his entire body as far distally as the ankles. He gave a history of troublesome nosebleeds several times weekly for as long as he could remember, one episode of tarry stools of two weeks' duration at age 14, and frequent bleeding from the gums. A sister had similar lesions but had no serious hemorrhage.

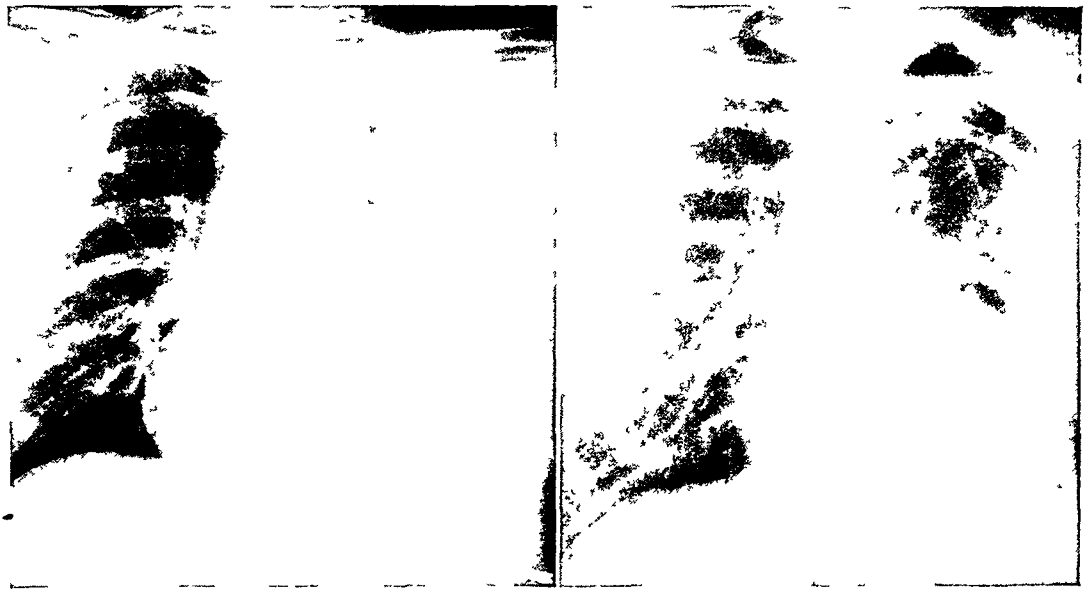

Fig. 3.—*A*, Roentgenogram after repeated thoracenteses, intercostal catheter drainage, and use of Varidase showing opacity of left chest with multiple air-fluid levels due to loculated hemothorax *B*, Roentgenogram over 7 months after decortication showing clearing of left hemithorax but the left diaphragm remains elevated and the costophrenic agle obliterated.

At thoracentesis on Feb. 15, 1952, 1,600 c.c. of blood was removed. He was given 1,000 c.c. of blood immediately, and 500 c.c. the following day. Bronchoscopy was negative. On the fourth day, a thoracentesis yielded 1,100 c.c. of blood and resulted in roentgen evidence of some clearing of the effusion but a day later there was a reaccumulation of the fluid (Fig. 2). On February 22, an intercostal catheter was inserted through the ninth intercostal space in the left posterior axillary line and 3,200 c.c. of blood removed. Thereafter, it drained about 500 c.c. of blood daily and he was supported by multiple transfusions. The intercostal catheter became plugged frequently and small amounts of Varidase were used on several occasions to clear it. This therapy was abandoned because it seemed to perpetuate the bleeding. X-rays, on February 29, showed a loculated hemothorax (Fig. 3).

On March 5, a left thoracotomy was performed and no evidence of tumor found. Decortication of the clotted hemothorax was carried out and it was noted that there was excessive bleeding from many tiny areas of the pleura. A total of 4,000 c.c. of blood was required during and immediately following surgery. At this time the bleeding time, clotting time, platelet count, clot retraction, prothrombin, Factor 5, and prothrombin utilization were normal Coombs tests direct and indirect were negative Fibrinolysin was negative after five and one half hours and a test for circulating anticoagulant was also negative

The patient drained large amounts of bloody fluid via the intercostal catheter but the lung failed to re expand completely On March 19, he began having a septic fever and a culture of the pleural fluid showed hemolytic staphylococcus On April 2, a rib resection was done and a large empyema pocket evacuated His course was one of steady improvement from that time on, and on May 20, he received his twenty fifth and final transfusion He had frequent expistaxis while in the hospital, but his chest symptoms gradually improved The lung slowly re expanded although he still had slight drainage at the time of his discharge He has been followed for three years and has had epistaxis and hematuria but no recurrence of hemothorax

DISCUSSION

In the case of spontaneous hemothorax reported by Armentrout and Underwood,[1] the diagnosis of hereditary telangiectasia is undeniable The patient at the age of 25 apparently had hemothorax because ‘ her physician found signs of fluid in her chest and aspirated blood from her left thorax in an amount estimated by her to be about one and one half quarts Convalescence from this illness was uneventful and she had no recurrence ” Our case is the second one of hemothorax reported in a patient known to have hereditary telangiectasia His prolonged illness is in marked contrast to the uneventful recovery of the patient just mentioned We now believe that the many small but vigorously bleeding areas encountered in the pleura at the time of decortication represented small telangiectases Extensive scarring prevented their definite identification as such at this time Prompt use of intercostal catheter water seal drainage with a slight negative pressure would be more likely to control the bleeding and prevent a clotted hemothorax than would repeated aspirations or surgical intervention We would be reluctant again to attempt decortication in such a patient during the active bleeding phase However, decortication was the logical treatment in this case because localized arteriovenous fistula or tumor could not be ruled out and there was present a partially clotted, loculated, infected hemothorax

There have been two reports of hemothorax from arteriovenous fistulas in patients who apparently showed no extrapulmonary lesions of hereditary telangiectasia[18] The telangiectases may have been unrecognized or considered unimportant One of these patients died at the age of 23 from hemothorax and was shown to have multiple pulmonary arteriovenous fistulas The other, reported by Bowers,[19] died of hemothorax on the second day of life and was found to have multiple pulmonary arteriovenous fistulas

Whether all pulmonary arteriovenous fistulas are manifestations of hereditary telangiectasia is open to question In many of the original descriptions of pulmonary arteriovenous fistulas, the peripheral lesions of hereditary telangiectasia are not mentioned They may have been unrecognized or considered irrelevant Only in recent years has the relationship between the two lesions been recognized Cardiologists and thoracic surgeons are now aware of the frequent coexistence of pulmonary arteriovenous fistulas and hemorrhagic telangiectasia. In most recent reports of pulmonary arteriovenous fistulas, note has been made that the patients were known to have hereditary telangiectasia.[13, 15, 17, 18, 20-26]

SUMMARY

This is the second reported case of spontaneous hemothorax in a patient with hereditary hemorrhagic telangiectasia. Treatment consisted of thoracentesis, intercostal catheter drainage with Varidase instillation, and later decortication. The likelihood of recurrent pleural hemorrhage from multiple small arteriovenous fistulas makes the treatment of hemothorax particularly difficult in this disease. Surgical intervention should probably be reserved as a last resort, or in the treatment of complications of a clotted hemothorax.

The occurrence of pulmonary arteriovenous fistulas in patients with hereditary telangiectasia is so common that chest x-rays should be a part of the routine work-up of these patients.

We suspect that hereditary hemorrhagic telangiectasia could be diagnosed in many cases of "spontaneous hemothorax" by a careful history and search for the lesions.

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MASSIVE CYSTIC PULMONARY HAMARTOMA

Report of Two Cases

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PULMONARY hamartoma has been found in 0.25 per cent of a series of consecutive necropsies.[1] Most of these tumors are small and are unrecognized in the ordinary roentgenogram of the thorax. Many of the smaller ones are interpreted as Ghon tubercles. When a hamartoma attains a size greater than 1 cm., it becomes a diagnostic problem. A patient with such a problem usually undergoes diagnostic thoracotomy. A hamartoma removed under these circumstances is seldom larger than 5 cm. in diameter. In two of the hamartomas of the lung recorded in the files of the Mayo Clinic, the most unusual feature was the large size. These two cases form the basis of this report.

BACKGROUND INFORMATION

In 1904, Albrecht[2] originated the term "hamartoma," derived from the Greek words meaning "error" and "tumor." Translated, his definition was "tumorlike malformations in which occur only an abnormal mixture of the normal constituents of the organ in which they are found. These malformations may consist of a change in the quantity, arrangement, degree of differentiation, or any combination of these. The conclusion to be drawn from histologic examination is that they originated in abnormal mixing of tissues during their development." Albrecht was referring to specific tumors of the spleen, liver, and kidney.

In 1922, Feller[3] initiated use of the term "hamartoma" in conjunction with certain benign cartilaginous pulmonary lesions, but only recently has the application of this term to these lesions been generally accepted. Until the past decade, most cases were recorded as chondroma of the lung, despite frequent recognition of their complex tissue structure since the nineteenth century.[1]

Pulmonary hamartoma occurs more frequently in males, in a ratio of 2 or 3 to 1. The range in age is from prematurity to old age, but those reported in childhood are few in number, probably because of the dearth of necropsy material and the relative infrequency of routine roentgenographic examina-

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tion of the thorax in this age group In a series of 156 indeterminate solitary circumscribed pulmonary lesions reported by Hood and co workers,[5] 16 per cent proved to be hamartomas No significant predilection for a given lobe or lung has been noted

Clinically, hamartomas are frequently asymptomatic and are discovered by routine x-ray examination Cough or thoracic pain may occasionally be the presenting symptom Endobronchial hamartomas commonly produce wheezing and symptoms of pulmonary suppuration Ten per cent are said to arise endobronchially, but this figure is derived from collected series and is undoubtedly influenced by the tendency to report lesions in unusual sites In our experience, true chondromas occur more commonly in this position than do pulmonary hamartomas

A roentgen shadow of a sharply circumscribed, peripheral pulmonary lesion with lobulated margins and clear adjacent pulmonary tissue should suggest the possibility of hamartoma The demonstration of diffuse punctate areas of calcification in such a lesion makes the diagnosis probable Lemon and Good[4] have emphasized the inadequacy of roentgenograms in establishing a definitive diagnosis, and we firmly believe that surgical exploration is indicated in all patients if their general condition permits

At thoracotomy, many hamartomas are readily recognizable in situ They are characteristically less than 4 cm in diameter, spherical or ovoid, subpleural, sharply demarcated from pulmonary parenchyma, of firm cartilaginous consistency, and have a white nodular surface Diagnosis should be made by frozen section technique, thereby enabling maximal conservation of functioning lung The majority are readily enucleated or removed by wedge resection Occasionally they lie deeply imbedded in hilar structures, necessitating more extensive resection Endobronchial lesions usually require major resection, although the surgeon should be alerted to the possibility of bronchotomy with conservative excision of the tumor and bronchial reconstruction, provided the associated suppurative changes are of a reversible nature Tissues of both mesodermal and endodermal origin are present in pulmonary hamartomas There is considerable variation in microscopic appearance Usually, hyaline cartilage is present in abundance, with intervening clefts lined by columnar or cuboidal epithelium which may be ciliated or mucus producing in part Fibrous and myxomatous connective tissue and fat are often present, and more rarely elastic or fibrocartilage or smooth muscle may be identified Calcified areas are frequently present in the cartilage, and occasionally bone formation occurs None of the aforementioned tissues need necessarily be present to make a diagnosis of hamartoma In some of these lesions, areas may be found which are remarkably similar to normal bronchi Derangement of architecture, overabundance of fat, and occasionally a suggestion of embryonal tissue will be noted, however This feature is illustrated in Case 2

CASE REPORTS

Case 1 —A 54 year old woman was referred for treatment of a pulmonary cyst She had complained of moderate exertional dyspnea for four years, and of mild orthopnea that

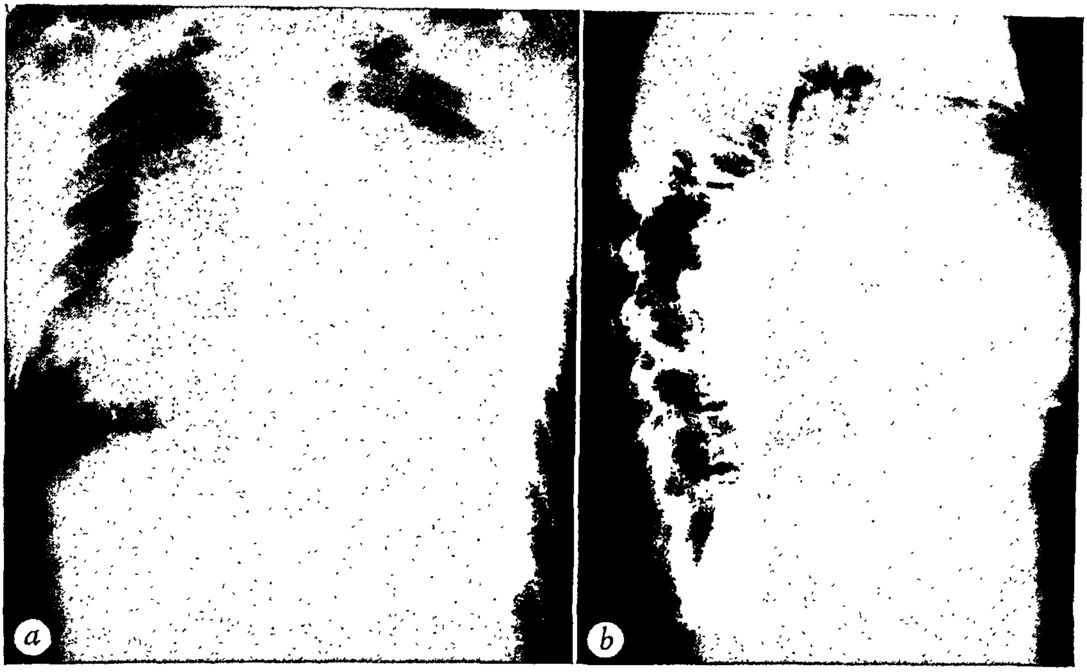

Fig. 1, *a* and *b*.—Case 1. Posteroanterior and left lateral roentgenographic views of the thorax showing hamartoma.

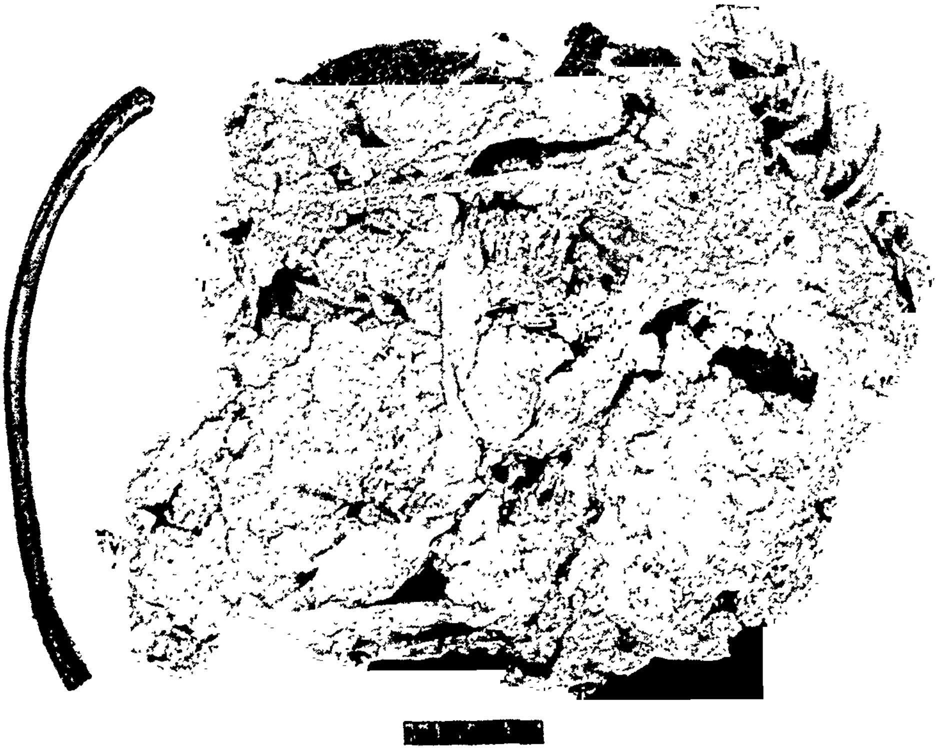

Fig. 2.—Case 1. Gross specimen of hamartoma with liquid contents evacuated.

was relieved by lying on her left side. Five times in the past three and one-half years her local physician had given her temporary relief by thoracentesis, which was productive of 1 to 1½ quarts of reddish-brown fluid.

Significant physical findings were the presence of all heart sounds to the right of the sternum and no evidence of functioning lung in the lower two-thirds of the left side of the thorax. The superficial veins of the neck were distended, particularly during inspiration. Laboratory data were within the normal range. Posteroanterior and lateral roentgenographic views of the thorax demonstrated a well-defined opacity in the lower three-fourths of the left hemithorax (Fig. 1). The heart and other mediastinal structures were markedly displaced to the right.

Left thoracotomy revealed a large, lobulated, cystic tumor occupying fully two-thirds of the left hemithorax. It involved most of the left lower lobe and was densely adherent to the pericardium, thoracic wall, lung, and diaphragm. During the extensive dissection necessary for removal of the lesion, its wall was ruptured, liberating 2,800 cc. of watery brown fluid. After repair of many small bronchial fistulas, the lung expanded well. Two thousand cubic centimeters of whole blood was given during the procedure. On the third postoperative day, coma developed and the neurological findings suggested a right cerebrovascular accident. Death occurred on the fourth postoperative day. Permission for necropsy was not granted.

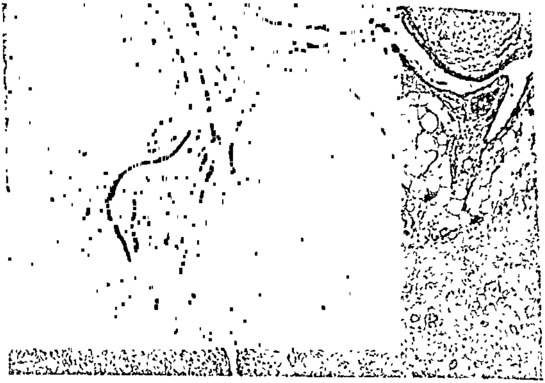

Fig. 3.—Case 1. Representative section from hamartoma. (Hematoxylin and eosin; ×50.)

The surgical specimen measured 17 by 15 by 12 cm. and weighed 600 grams without its fluid contents (Fig. 2). The surface was nodular and grayish white. A large single cyst occupied the center of the mass. The wall varied from 0.4 to 5.5 cm. in thickness and its inner surface was partly smooth, but there were many roughened areas where the tissue appeared to be necrotic. On cut surface the tumor appeared grossly to consist of hyaline cartilage with small scattered areas of dense calcification.

Microscopically, there was abundant hyaline cartilage with some areas of calcification (Fig. 3). Much adult fat and fibrous connective tissue were present and there were small cystic spaces lined with flattened and cuboidal epithelium. Part of the tissue was necrotic, and the presence of hemosiderin suggested previous hemorrhage. The large cyst was lined with cuboidal epithelium in the smooth areas.

Case 2.—A 27-year-old man was admitted for evaluation of a pulmonary cyst known to have been present for eleven years. He denied having any pulmonary symptoms. No unusual findings, except decrease in the breath sounds over the right part of the thorax, were revealed by a thorough physical examination.

Laboratory data were normal. Posteroanterior and lateral roentgenographic views of the thorax demonstrated a well-circumscribed cystic lesion occupying much of the right lower lobe (Fig. 4). A considerable amount of calcareous material was seen within the cystic lesion and also in the supradiaphragmatic region and along the thoracic wall below and lateral to the cyst. No bronchovascular markings were seen in the latter area.

Right thoracotomy disclosed a tumor replacing most of the right lower lobe. Resection of the lobe was accomplished without difficulty and the patient recovered uneventfully.

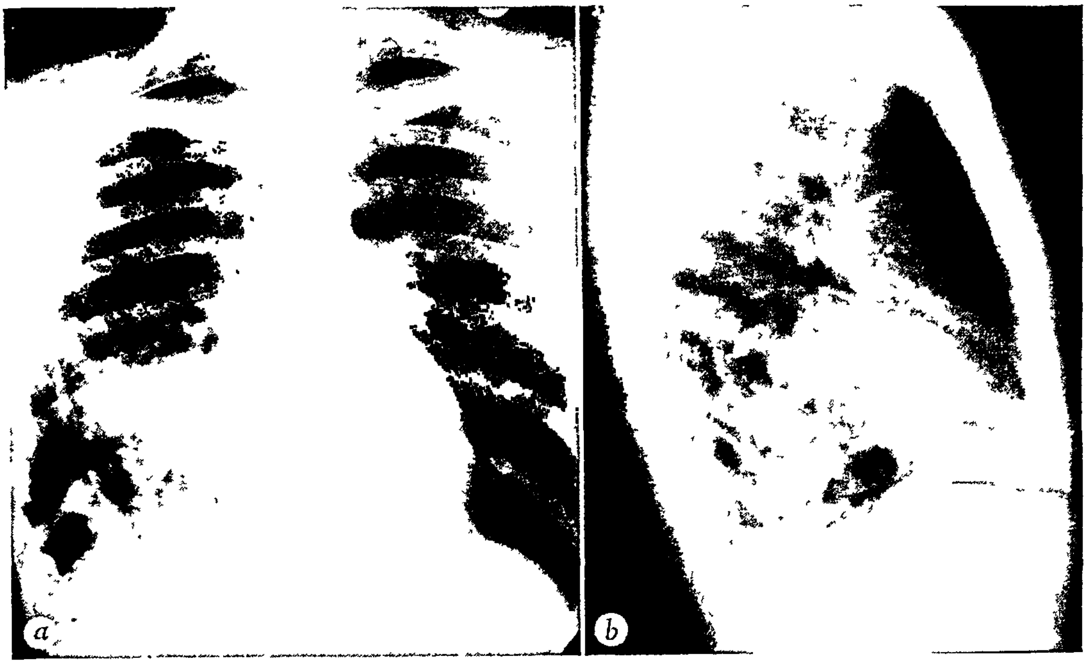

Fig. 4, *a* and *b*.—Case 2. Posteroanterior and right lateral roentgenographic views of hamartoma.

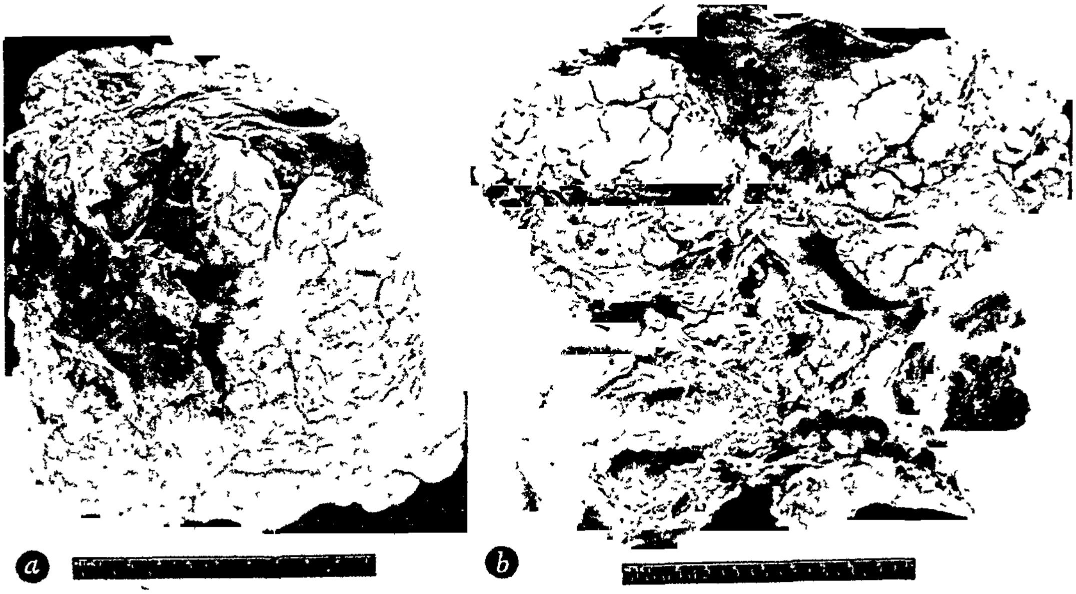

Fig. 5.—Case 2. *a*, Gross specimen removed at right lower lobectomy. *b*, Cross section illustrating multiloculated air-containing cysts.

The surgical specimen measured 16 by 14 by 10 cm. and weighed 800 grams (Fig. 5). It was bluish white and its surface was bosselated except for the undersurface, which was smooth and had the conformation of the diaphragm. The upper margin was covered by a layer of residual pulmonary tissue which stripped readily from the tumor. Many parts were of bony consistency but others were readily cut and had the appearance of hyaline cartilage. Small fluid-containing clefts were numerous. There were large multiloculated air containing cysts in its center. Continuity with the normal bronchial system could not be demonstrated.

Microscopically, large islands and connecting strands of hyaline cartilage encircled by fibrous connective tissue predominated in the field (Fig. 6). Between these areas were cystic spaces of variable size lined with cuboid, columnar, pseudostratified columnar and occasionally mucus-producing epithelium. The cartilage was calcified in many areas. Mature fat and bone made up a lesser part of the lesion, and myxomatous connective tissue, smooth muscle, and mucosal glands were also identified. The large air containing cysts were lined with columnar epithelium. Some areas strikingly resembled normal bronchi (Fig. 6, *b*). No alveolar structure was found within the confines of the tumor.

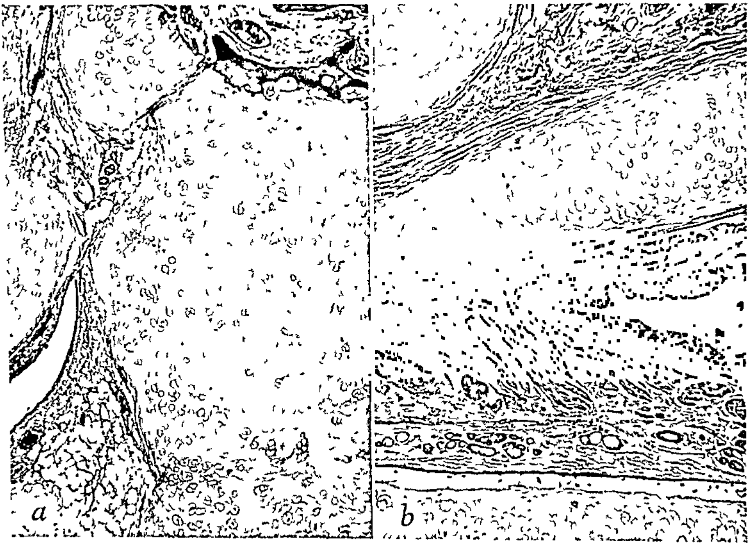

Fig 6.—Case 2. *a*, Section showing typical pattern of the bulk of the lesion. (Hematoxylin and eosin, ×30.) *b*, Section showing a closer resemblance to normal bronchi, with a high degree of differentiation of tumor tissue (Hematoxylin and eosin; ×40.)

In reviewing the literature, we found that a pulmonary hamartoma greater than 5 cm. in diameter is a medical rarity. Lesser, Linser, and Reboul,[7-9] each reported a case of benign hamartoma that measured in excess of 10 cm. in diameter. Schumann[10] described a hamartoma the "size of a man's head"; this tumor weighed 12 pounds (5.4 kilograms). The lesion reported by Busse[11] was huge, filling the entire hemithorax and exerting enough pressure to bulge the intercostal spaces. Careful analysis of his paper leaves considerable doubt as to the true nature of the tumor.

The premature infant reported by Jones[12] is of particular interest. Although the lesion measured only 3.5 cm. in diameter, it replaced the major portion of the right upper lobe. Cartilage and bronchial epithelium were present in small nests within islands of fibrous connective tissue separated by normal lung. Liebow[13] has referred to this lesion as a "diffuse hamartoma." We have seen no counterpart of it in adults and it seems reasonable to assume that, with growth of the lesion, the included pulmonary tissue could either be rendered functionless and made cystic or be overgrown to such an extent that it would be unrecognizable. If the growth differential is primarily in favor of the cartilage and epithelium in these malformations, the end result in Jones' case would have been identical to that in the 2 cases we have presented. Unfortunately, we have been unable to find another reported case of true hamartoma in an infant and, until these are available, the early development of hamartoma will remain in doubt.

The cystic nature of hamartomas deserves comment. Microscopic epithelium-lined clefts are characteristic of the lesions. The presence of small, grossly visible cysts is not unusual. McGlumphy[14] reported a hamartoma, 2 cm. in diameter, that contained a cyst 9 mm. in diameter. We did not find a case of benign pulmonary hamartoma with a large cyst or cysts in our review of the literature. A cystic component in these lesions may be suggested occasionally on the roentgenograms, but these "rarefied areas" usually prove to be fat tissue in the specimen. In both of the cases reported here, the masses contained large cysts and presented as cystic lesions clinically, one on the basis of roentgen findings and the other on the basis of a history of recurrent aspirations that were productive of large quantities of fluid.

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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

THIRTY-SIXTH ANNUAL MEETING

MAY 7, 8, AND 9, 1956

HOTEL FONTAINEBLEAU, MIAMI BEACH, FLA.

Program

MEMORANDA FOR GUIDANCE OF ESSAYISTS AND DISCUSSANTS

1. In accordance with the By-Laws of the Association the papers which are read at the Meeting shall be given to the Secretary *immediately after presentation. If these papers are not submitted promptly their publication may well be* delayed.
2. It is customary to group all of the discussions together on a series of papers. Any delay in the return of corrected discussions to the Editor means that publication of all papers on the subject will be held up. This is manifestly unfair to those who are conscientious in the prompt submission of their remarks for publication. Undue tardiness may mean that the discussion will not be published.
3. *Regular Program.*—Essayists (except in symposia) will be limited to 15 minutes for presentation.
4. *Thoracic Surgical Forum.*—Essayists will be limited to 10 minutes for their presentations. Papers on the Thoracic Surgical Forum as submitted for publication will be limited to 3,000 words. At the discretion of the President and within the limits of the allotted time, one or more discussions per paper will be allowed following all formal presentations.
5. *Discussants.*—Discussants will be limited to 3 minutes. Only members of the Association and Invited Speakers will have the privilege of discussing papers.

Monday Morning, May 7, 1956

8:30 A.M. Business Meeting.

9:00 A.M. Scientific Session: REGULAR PROGRAM.

1. **Congenital Diaphragmatic Hernia and Hypoplastic Lung: A Clinical Evaluation.** Benson B. Roe, San Francisco, Calif.
2. **Critically Crushed Chests: A New Method of Treatment by Internal "Pneumatic" Stabilization With Continuous Mechanical Hyperventilation.** Edward E. Avery (by invitation), E. Trier Mörch (by invitation), and Donald W. Benson (by invitation), Chicago, Ill.
3. **Complete Traumatic Rupture of the Left Main Bronchus Successfully Repaired Eleven Years After Injury.** Daniel E. Mahaffey (by invitation), Oscar Creech, Jr., Hollis G. Boren (by invitation), and Michael E. DeBakey, Houston, Texas.
4. **Congenital Atresia of Esophagus.** George H. Humphreys, Bruce M. Hogg (by invitation), and Jose Ferrer (by invitation), New York, N. Y.
5. **Torek Esophagectomy: The Case Against Segmental Resection for Esophageal Cancer.** William L. Watson, John T. Goodner (by invitation), Theodore P. Miller (by invitation), and George T. Pack (by invitation), New York, N. Y.

6. **Cardiac Surgery With Hypothermia and Acetyl Choline Arrest.** Peter V. Moulder (by invitation), Richard Thompson (by invitation), Curtis Smith (by invitation), and William E. Adams, Chicago, Ill.

Monday Afternoon, May 7, 1956

2:00 P.M. Scientific Session: REGULAR PROGRAM.

Symposium on Anesthesia and Related Problems (papers 7-11 inclusive)

7. **Contrast of Two Systems of Anesthesia for Thoracic Surgery.** Henry Beecher, Boston, Mass.
8. **A Consideration of Proper Methods in Thoracic Anesthesia.** E. M. Papper, New York, N. Y.
9. **The Cardiorespiratory Dynamics of Controlled Respiration in the Open and Closed Chest.** Archer S. Gordon (by invitation) and Charles W. Fyre (by invitation), Chicago, Ill.
10. **The Significance of the Lung-Thorax Compliance in Ventilation During Thoracic Surgery.** William E. Brownlee (by invitation) and Frank F. Allbritten, Jr., Kansas City, Kansas.
11. **Inefficient Carbon Dioxide Absorption Requiring Increased Pulmonary Ventilation During Operations With an Open Thoracotomy.** Thomas F. Nealon (by invitation), George J. Haupt (by invitation), Harold Chase (by invitation), and John H. Gibbon, Jr., Philadelphia, Pa.

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12. **Aortic Valvulotomy Under Direct Vision During Hypothermia.** F. John Lewis, Norman E. Shumway (by invitation), Suad A. Niazi (by invitation), and Robert B. Benjamin (by invitation), Minneapolis, Minn.
13. **The Indications for Lobectomy in the Treatment of Carcinoma of the Lung.** Joseph L. Robinson and John C. Jones, Los Angeles, Calif.
14. **Common Factors in Lung Cancer Survivors.** Richard H. Overholt and James A. Bougas (by invitation), Boston, Mass.

6:30 P.M.—8:30 P.M. COCKTAIL PARTY. Informal. Hotel Fontainebleau.

Tuesday Morning, May 8, 1956

8:30 A.M. Scientific Session: THORACIC SURGICAL FORUM.

15. **Introducing a Simple Surgical Method for the Correction of Mitral Regurgitation Using the Finger Ring Valve Elevator.** Aran S. Johnson (by invitation). Sponsored by William M. Tuttle, Detroit, Mich.
16. **Further Experiences With the Method of Controlled Unilateral Pulmonary Artery Occlusion in the Study of Lung Function.** Paul Nemir, Jr., H. H. Stone (by invitation), H. R. Hawthorne (by invitation), and T. N. Mackrell (by invitation), Philadelphia, Pa.
17. **Oxygen Availability to the Brain During Inflow Occlusion of the Heart in Normothermia and Hypothermia.** Samuel Kaplan (by invitation), Edward C. Matthews (by invitation), Louis Schwab (by invitation), and Leland C. Clark (by invitation). Sponsored by James A. Helmsworth, Cincinnati, Ohio.
18. **The Effect of Somatotrophin on Ventricular Fibrillation of Arterioclusive and Hypothermic Origin.** David H. Watkins (by invitation), S. Rothman (by invitation), Arthur E. Prevedel (by invitation), and Gordon A. Munro (by invitation). Sponsored by William B. Condon, Denver, Colo.
19. **Physiologic Responses in Man to Total Body Perfusion for Open Intracardiac Surgery.** Richard A. DeWall (by invitation) and Raymond C. Read (by invitation), Minneapolis, Minn.

20. **A Method for Controlled Cardiac Arrest as an Adjunct to Open Heart Surgery.** W. Glenn Young (by invitation), Will C. Sealy, Ivan W. Brown, Jr. (by invitation), Wilmer C. Hewitt, Jr. (by invitation), Henry A. Callaway, Jr. (by invitation), Doris H. Merritt (by invitation), and Jerome S. Harris (by invitation), Durham, N. C

21. **The Neutralization of Heparin by Protamine in Extracorporeal Circulation.** Mr. Raymond Hurt (Travelling Fellow of the Association) (by invitation), London, England.

22. **Experience With a Disposable, Artificial Lung.** D B. Effler, W. J. Kolff (by invitation), L. K Groves (by invitation), F. M. Sones, Jr. (by invitation), and G. Peereboom (by invitation), Cleveland, Ohio.

23. **An Artificial Lung Dependent Upon Diffusion of Oxygen and Carbon Dioxide Through Plastic Membranes.** George H A Clowes, Jr, Amos L Hopkins (by invitation), and William E Neville (by invitation), Cleveland, Ohio.

24. **Extracorporeal Circulation for Open Heart Surgery.** Russell M. Nelson (by invitation), Hans H. Hecht (by invitation), Richard W Hardy (by invitation), and Joe Burge (by invitation), Salt Lake City, Utah

25. **A Mechanical Pump Oxygenator for Direct Vision Repair of Atrial Septal Defects.** Bernard S. Levowitz (by invitation), Melvin M Newman, Jackson H Stuckey (by invitation), Marie C. Kernan (by invitation), Harry N. Iticovici (by invitation), and Clarence Dennis, Brooklyn, N. Y.

26 **Elimination and Transport of Mucus in the Lung: An Experimental Study in the Dog.** Edward G. Huppler (by invitation), O Theron Clagett, and John H. Grindlay (by invitation), Rochester, Minn

27 **Surgical Treatment of Ventricular Septal Defects: A Method Utilizing Selective Coronary Perfusion and Hypothermia.** John B. Grow, Charles V. Demong (by invitation), and Charles R Hawes (by invitation), Denver, Colo

28. **A Report on Heterologous Vascular Shunts (Bovine Brachiocephalic) in Experimental Aortic Arch Resection.** Ralph D. Alley, William H Sewell (by invitation), Allan Stranahan, Harvey W Kausel (by invitation), Thomas S Reeve (by invitation), and Alan S Peck (by invitation), Albany N. Y

29. **Arterial Homograft Substitution and Bypass in Superior Vena Caval Obstruction.** John F Higginson, Portland, Ore

30 **Experiments With Substitute Esophagus.** Laurence Rubenstein (by invitation), Chicago, Ill

Tuesday Afternoon, May 8, 1956

2 00 P M Executive Session (Limited to Active and Senior Members)

3 00 P M. Scientific Session · REGULAR PROGRAM

Address of the President, Richard H Meade, Grand Rapids, Mich
Some of the Forgotten Men in the Field of Thoracic Surgery.

Address of Honored Guest, Mr. Philip Allison, Ch M , F R C S , Nuffield Professor of Surgery, Oxford, England.
Observations on a Conservative Approach to Nonmalignant Lesions at the Cardia.

31. **Reconstruction of the Trachea.** John M. Keshishian (by invitation), Brian Blades, and Edward J Beattie, Jr , Washington, D C

32 **The Solitary Pulmonary Nodule—A Ten-Year Survey.** Edgar W Davis, J Winthrop Peabody, Jr (by invitation), and Sol Katz (by invitation), Washington, D. C.

7:00 P.M. COCKTAILS, BANQUET AND DANCING, Hotel Fontainebleau.
Attendance limited to members of the association and their ladies, invited speakers and their ladies.
Dinner dress preferred.

Wednesday Morning, May 9, 1956

9:00 A.M. Scientific Session: REGULAR PROGRAM.

33. **Pulmonary Resection for Tuberculosis: A Five- to Ten-Year Follow-up Study.** James D. Murphy and James M. Davis (by invitation), Oteen, N. C.
34. **The Role of the Chronic Occult Postresection Bronchial Fistula in the Reactivation of Tuberculosis: Pathogenesis and Treatment.** John W. Bell (by invitation) and E. M. Medlar (by invitation), Sunmount, N. Y.
35. **Extraperiosteal Plombage Thoracoplasty. Operative Technique and Results With 161 Cases With Unilateral Surgical Problems.** Norman J. Wilson, Orlando Armada (by invitation), William V. Vindzberg (by invitation), and William B. O'Brien (by invitation), Boston, Mass.
36. **Bronchographic Studies as a Guide in the Surgical Treatment of Pulmonary Tuberculosis.** F. J. Phillips, Anthony Lalli (by invitation), and Walther Buhler (by invitation), Bartlett, Alaska.
37. **A Clinical Evaluation of Decortication.** David H. Waterman, Sheldon E. Domm, and William K. Rogers (by invitation), Knoxville, Tenn.

Wednesday Afternoon, May 9, 1956

2:00 P.M. Scientific Session: REGULAR PROGRAM.

38. **Observations on the Surgical Pathology of Congenital Intracardiac Defects Made During Direct Vision Repair.** Herbert E. Warden (by invitation), Richard L. Varco, and C. Walton Lillehei, Minneapolis, Minn.
39. **Technique for Repair of Ventricular Septal Defects Utilizing Extracorporeal Circulation.** John W. Kirklin, H. G. Harshbarger (by invitation), D. E. Donald (by invitation), and J. E. Edwards (by invitation), Rochester, Minn.
40. **The Use of the Heart-Lung Apparatus in Human Cardiac Surgery.** F. D. Dodrill, Norman Marshall (by invitation), Jan Nyboer (by invitation), Elsie Noe (by invitation), C. H. Hughes (by invitation), and A. B. Stearns (by invitation), Detroit, Mich.
41. **The Treatment of Mitral Insufficiency by the "Purse-String" Technique.** Robert P. Glover, Julio C. Davila (by invitation), and O. Henry Janton (by invitation), Philadelphia, Pa.
42. **Mitral Insufficiency—Treatment by Polar Cross-Plication of the Annulus Fibrosus.** Henry T. Nichols (by invitation), Philadelphia, Pa.
43. **Patent Ductus Arteriosus in Infancy.** S. Richard Bauersfeld (by invitation), Paul C. Adkins (by invitation) and Edward M. Kent, Pittsburgh, Pa.

The Journal of
Thoracic Surgery

Vol. 31 May, 1956 No. 5

Original Communications

THE OSTEOPLASTIC WALL THORACOPLASTY AFTER PNEUMONECTOMIES

Viking Olov Björk, M.D.
Stockholm, Sweden

THE PRINCIPLE of simultaneous pneumonectomy and thoracoplasty is not new. As early as 1933, when Evarts Graham performed the historic pneumonectomy for carcinoma of the lung, he completed this operation with a resection of ribs three to ten. Graham continued to use simultaneous thoracoplasty in the first 5 or 6 cases of pneumonectomy but then left this practice as a routine. In cases with pneumonectomy for tuberculosis, however, he continued to perform a simultaneous thoracoplasty in order to diminish the possibility of tuberculous empyema. Later Iverson, and Skinner,[6] 1950, Conklin, Tuhy, and Grismer,[4] 1951, and Samson,[7] 1951, advocated thoracoplasty at the same stage as pneumonectomy for tuberculosis.

The name thoracoplasty means to mold the chest. Earlier techniques, however, have mainly consisted of rib resection. A stable chest wall is then only obtained when the rib regeneration has occurred. This new chest wall may have the desired size and shape, but will be greatly influenced by the scapula and the re-expanding lung.

As the name thoracoplasty is generally accepted for any kind of rib resection, the name osteoplastic has been added to the procedure I have devised to diminish and mold the chest into a desired shape. The principle is to resect posterior ends of the ribs and bend them in the costal cartilage to form a new roof of the chest. The ribs are fixed posteriorly by nylon or stainless steel sutures through drill holes to the posterior end of the uppermost intact rib. This osteoplastic thoracoplasty is then sized according to the amount and quality

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From Sabbatsberg Hospital, Stockholm, Sweden, Surgeon-in-Chief: C. Crafoord, M.D., Professor.

This work was aided by The Swedish National Association Against Tuberculosis.
Received for publication May 22, 1955.

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of the remaining lung tissue at the end of the pulmonary resection for tuberculosis. Good experience with the osteoplastic roof thoracoplasty, now performed in more than 200 cases since 1953, has made me try to utilize the same principle after pneumonectomies.

EARLIER METHODS

1. *Pneumothorax.*—After a pneumonectomy for tuberculosis, the remaining space may be left to become obliterated without thoracoplasty. Especially on the left side where the elevated diaphragm can significantly reduce the space, the herniation of the other lung, with increasing emphysema, may not be so pronounced. Air refills in the remaining space will keep the mediastinum in the mid-line for some time and prevent a too rapid overexpansion of the remaining lung.

To follow this line of treatment the following two criteria must be fulfilled: (a) possibilities for infection in the remaining space must be excluded; and (b) no demonstrable tuberculosis should be found in the remaining lung.

In our material, both these criteria will very seldom be fulfilled at the same time.

2. *Rib resection.*—The most common method of diminishing the thoracic cavity after a pneumonectomy is by rib resection. This will then mean a two-stage procedure. Usually the lung is removed first and then the ribs later on. The simultaneous rib resection has been performed but is not recommended, especially not after an extrapleural resection, where only ribbons of periosteum and intercostal muscles remain of the chest wall after a rib resection. The paradoxical movements of the mediastinum will make it difficult for the patient to raise his sputum, although the procedure is tolerated by good risk patients.

Earlier, we often performed first a five-rib thoracoplasty, which then at a second stage was extended, when the lung was removed. The rib regeneration after the thoracoplasty, however, often made it difficult to obliterate effectively the remaining space after the pneumonectomy in the second stage. Most of these cases therefore have a considerable herniation of the remaining lung into the side upon which the operation had been performed.

3. *Plombage.*—Plastic material to fill the space is now rarely used. This method has caused too much trouble with infection and cavity perforations, when used over lung tissue. When used to fill a space after total pneumonectomy, either intrapleurally (7 cases) or extraperiosteally (5 cases), we have so far not encountered any complications. Only patients with a healthy pleura were chosen in whom there was thought to be no danger of infection.

In my series of 155 cases of pneumonectomies for tuberculosis, I have thus only used plombage to obliterate the space in 12 cases.

THE AUTHOR'S METHOD: AN OSTEOPLASTIC WALL THORACOPLASTY

Indications.—There is always great trauma for the patient, to wake up after a major intrathoracic operation only to look forward to another operation in the near future. Besides the psychologic trauma, the stay in the hospital is prolonged. Furthermore, it is more difficult to regain full arm movements and obtain a perfect healing of the muscles after a second operation.

The osteoplastic wall thoracoplasty after a pneumonectomy gives the patient all the advantages of a one-stage procedure. The immediate obliteration of the remaining space will diminish the incidence of infection. Therefore, in cases of pneumonectomy for tuberculosis, especially in cases of extrapleural resection due to empyema, the operation is completed with an osteoplastic wall thoracoplasty. In 1 case where a tuberculous empyema was in the state of breaking through the chest wall, the space got infected after resection with osteoplastic thoracoplasty. All of the chest wall except the skin had to be resected. In such a case, it is better to resect the whole chest wall together with the lung at once, utilizing prolonged artificial ventilation for the postoperative treatment.

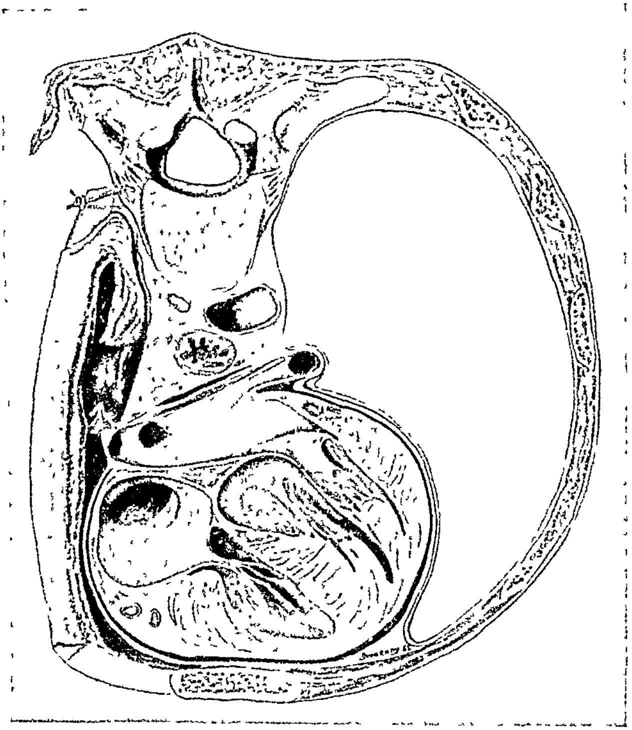

Fig. 1.—The ribs are resected posteriorly. They are then bent or fractured in the costochondral junction, so they lie against the mediastinum. The posterior ends are fixed with a nylon suture through a drill hole in the rib and through the costovertebral ligament.

Technique.—In cases with an accumulation of secretion behind a bronchial stenosis or with empyema and bronchopleural fistula, it is essential to prevent spill over. A left-sided bronchial stenosis will prevent the use of endobronchial anesthesia with a Carlens tube and then, too, the face-down position, recommended by Overholt, is the position of choice. When one is familiar with the prone position, it will be *preferred for all pneumonectomy* cases.

In a case with a thick-walled empyema, it is sometimes preferable to do the extrapleural "strip" from the chest wall with the ribs in place. Often some hard work may, however, be saved by first resecting the posterior ends of the upper *9 ribs on the left side* and the upper *10* ribs on the right side from the tip of the transverse process. After completion of the pneumonectomy, the osteoplastic wall thoracoplasty is carried out in the following way.

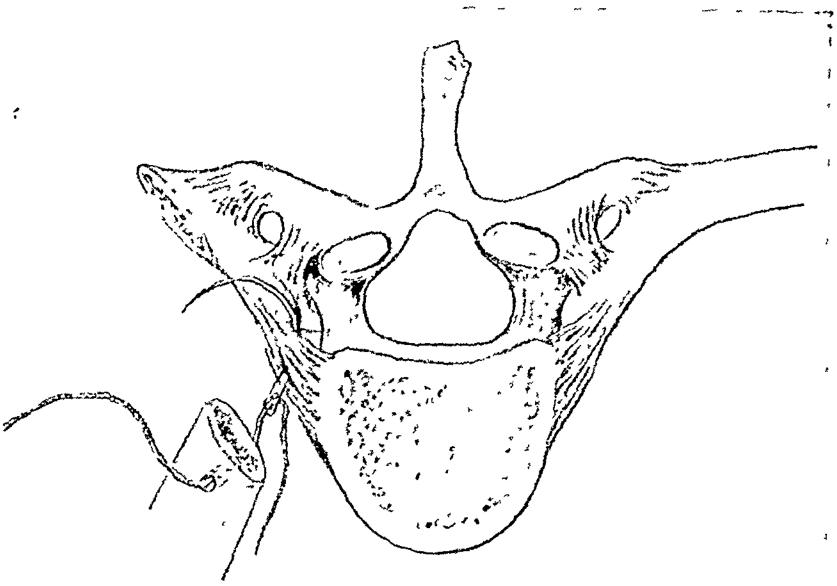

Fig. 2.—The posterior ends of the ribs are fixed to the vertebral body by a nylon suture through the costovertebral ligament and then through a drill hole in the rib.

Rib resection:

a. The entire length of the first rib, including its costal cartilage, is resected to prevent a remaining apical air space. The first rib has only been left in place in absolutely clean cases.

b. Ribs 2 to 9 on the left side and 2 to 10 on the right side are shortened *enough to become* straight, so that when they are bent or preferably fractured in the costochondral junction, they will reach the vertebral body (Fig. 1).

c. A drill hole (an especially devised drill bore manufactured by Stille, Sweden, is used) is then made in the posterior end of the ribs. The ribs are then fixed to the vertebral bodies by heavy nylon sutures through the drill holes in the ribs, usually through the costovertebral ligament (Fig. 2).

In the beginning, the posterior ends of the ribs were sutured to the anterior longitudinal ligament of the spine but a much stronger fixation is usually obtained by placing the nylon sutures through the costovertebral ligament and *behind the tip of the head of a rib* (Figs. 1 and 2).

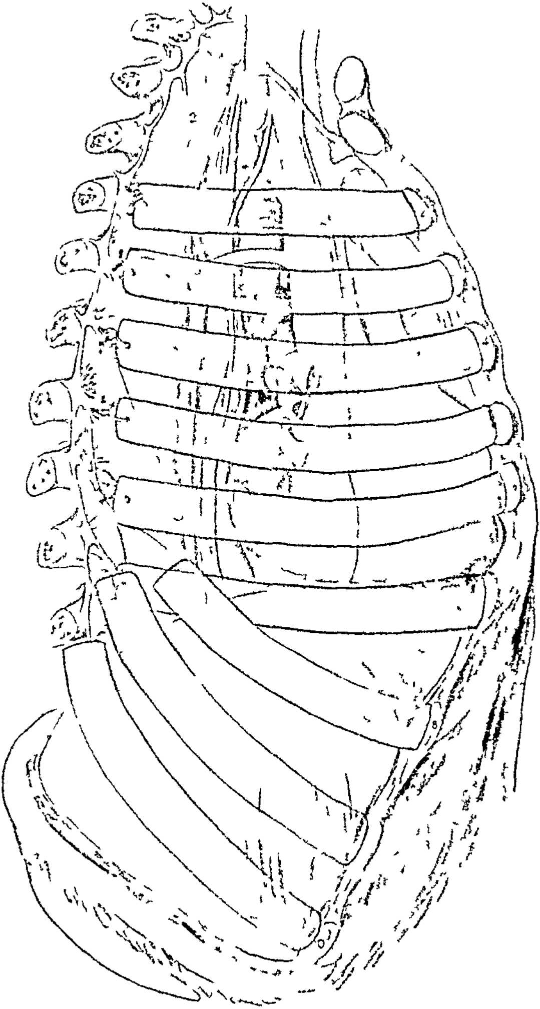

Fig 3 —The first rib is resected Then ribs 2 to 7 are bent down and fixed to the thoracic vertebrae 4 to 9 Ribs 8 to 9 on the left side and 8 to 10 on the right side are resected posteriorly but left outside the fixed ribs to round off the contour

Rib 2 is fixed to the fourth thoracic vertebra.
Rib 3 is fixed to the fifth thoracic vertebra.
Rib 4 is fixed to the sixth thoracic vertebra.
Rib 5 is fixed to the seventh thoracic vertebra.
Rib 6 is fixed to the eighth thoracic vertebra.
Rib 7 is fixed to the ninth thoracic vertebra (Fig. 3).

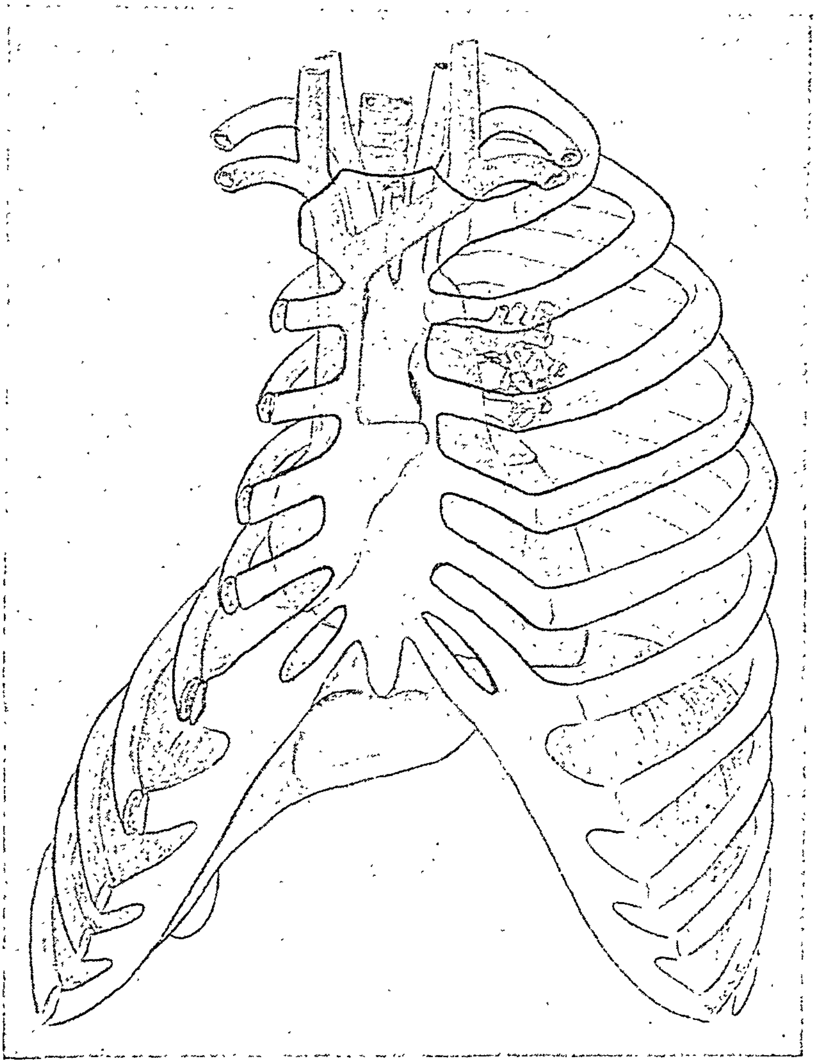

Fig. 4.—A stable wall for the mediastinum is obtained. The space will be obliterated and a herniation of the remaining lung prevented.

When rib 7 or sometimes 8 has been fixed to the lowest possible thoracic vertebra, a stable wall for the mediastinum is obtained (Figs. 1 and 4). All nylon sutures are first placed before they are tied. All intercostal nerves below the fifth intercostal space are left intact as the nylon sutures may pass between the intercostal muscle bundles. The two or three lowermost resected ribs do not have to be fixed but are left outside the fixed ribs to round off the contour (Fig. 4). One tube for drainage is left inside the ribs for two to three days (Figs. 5 through 9, Cases 1 to 3).

With this technique the operation is usually prolonged only by about one-half hour The operation is well tolerated also in cases with considerable ventilatory insufficiency, provided artificial ventilation is maintained during the first postoperative period and until the patients have regained strength enough for coughing and spontaneous ventilation (Figs 7 and 8, Case 2) Twenty patients altogether have had a pneumonectomy and simultaneous osteoplastic wall thoracoplasty performed Three patients are dead 1 after three weeks due to infection with bronchial fistula, 1 after ten days in bronchopneumonia on the remaining lung (autopsy showed a hematoma on the operated side but no source of bleeding) The third patient died six months after the operation in cor pulmonale with pulmonary edema

When a pneumonectomy is performed under an old thoracoplasty, it may be difficult to obliterate the space by extended rib resection The same principle may then be followed First, the posterior part of the chest wall with rib regenerations is excised The regenerated ribs should then not be removed one by one but the whole part of the chest wall excised in one block The anterior parts of the ribs are fractured in the costochondral junction and fixed to the vertebral body in the way just described

In cases without danger of infection in the remaining space, I have tried to resect the posterior part of rib 2, bent it down and fixed it to the transverse process of the sixth vertebra in order to keep the trachea and the mediastinum in the mid line Then rib 4 was resected posteriorly and bent down around the heart and fixed to the transverse process of the ninth vertebra One year after this operation on a 23 year old man, a considerable herniation of the remaining right lung had occurred (Case 4, Fig 10), although the trachea was only slightly displaced to the left The functional result was very good, maximal breathing capacity was 92 L per minute and the residual air was only 22 per cent of the total volume The cosmetic result was excellent As it is essential in most cases to obliterate the space to prevent infection and to prevent herniation of the remaining lung, this modification is now rarely used The deformity caused by the osteoplastic wall thoracoplasty has to be accepted if the space is to be obliterated and herniation completely prevented See Fig 11, *A*, *B*, and *C* which show a 30 year old woman, one year after an extrapleural pneumonectomy with osteoplastic wall thoracoplasty on the right side

CASE REPORTS

Case 1—A 46 year old woman was found to have tuberculous infiltration in the right upper lobe in 1952 and complete atelectasis of the right lung in 1953 The right lung became destroyed behind a stenosis of the right main bronchus (Fig 5) At operation a right extrapleural pneumonectomy was performed and simultaneously an osteoplastic wall thoracoplasty (Fig 6)

Case 2—A 40 year old woman had had tuberculous infiltrations in both upper lobes since 1926 A pleurisy on the right side in 1949, resulted in an empyema that persisted for six years There was bilateral cavitation (Fig 7) *Before operation*, the arterial oxygen tension at rest was 55 mm Hg The maximal breathing capacity was 23 L per minute Seventeen per cent of the oxygen uptake and 23 per cent of the ventilation were on the right side The patient withstood a right extrapleural pneumonectomy with osteoplastic wall thoracoplasty very well with the aid of prolonged artificial ventilation, and is in a good condition ten months later (Fig 8)

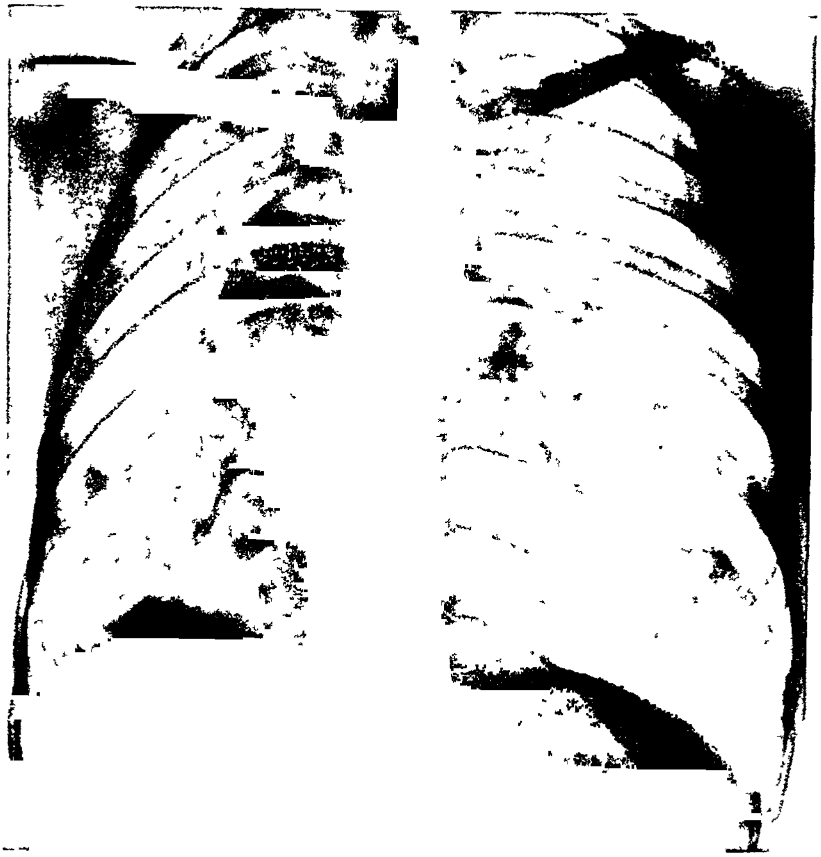

Fig. 5.—Case 1. An x-ray of a 46-year-old woman with stenosis of the right main bronchus and a destroyed right lung.

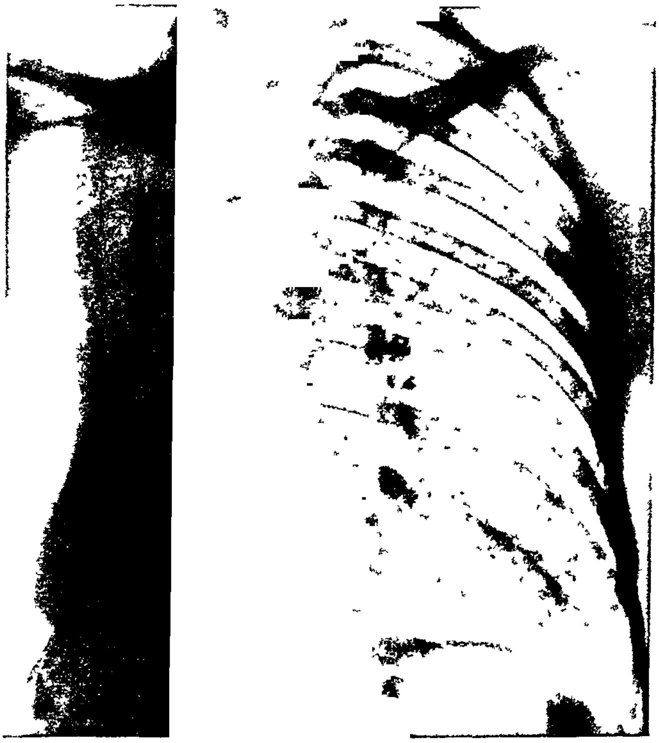

Fig. 6.—Case 1. An x-ray taken three months after an extrapleural pneumonectomy and osteoplastic wall thoracoplasty on the right side.

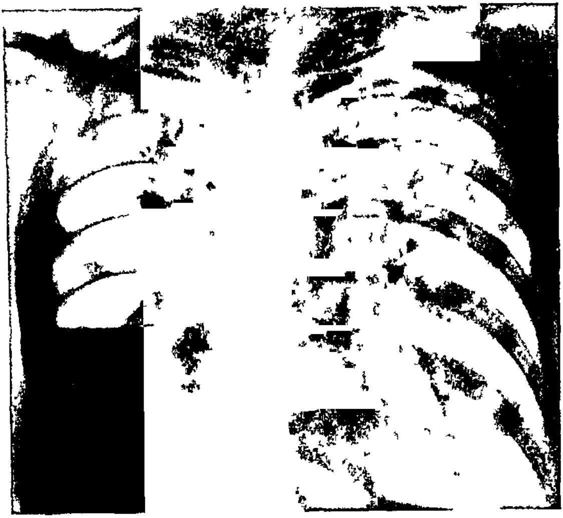

Fig 7—Case 2 An x ray of a 40 year old woman with bilateral cavitary tuberculosis and a right sided empyema of six years duration Maximal breathing capacity 23 L per minute Seventeen per cent of the total oxygen uptake and 23 per cent of the ventilation came on the right side

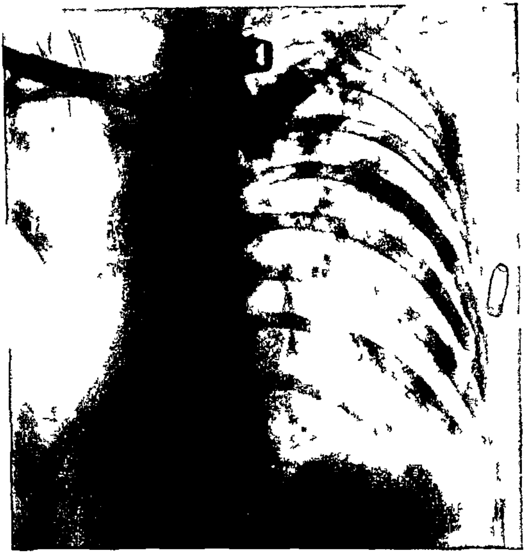

Fig 8—Case 2 X ray taken the day after a right extrapleural pneumonectomy and an osteoplastic wall thoracoplasty Artificial ventilation was maintained for two weeks Two days after operation the patient took over spontaneous ventilation for one hour and then for gradually increasing lengths of time After three weeks, she could walk slowly around in the ward and is in good condition ten months later

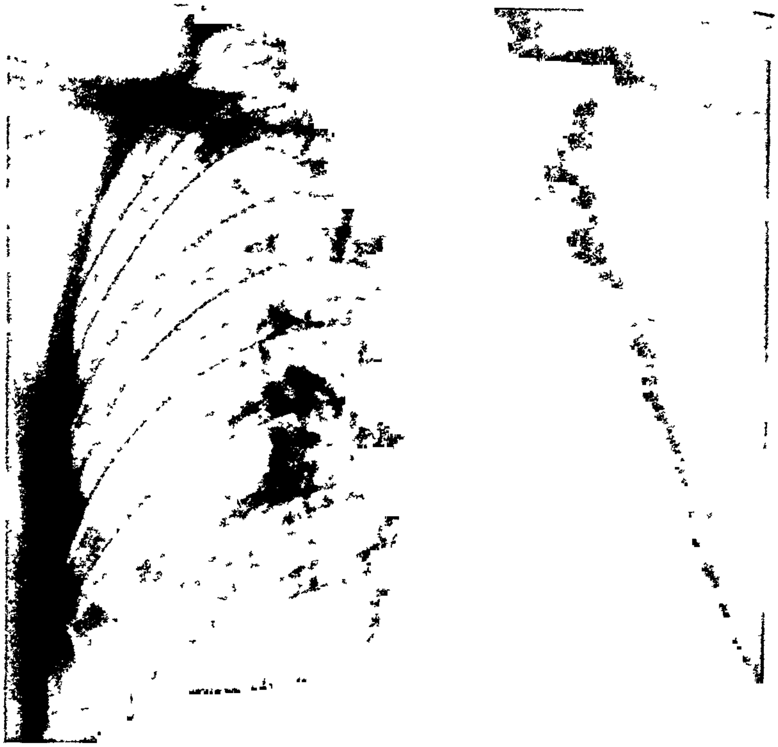

Fig. 9.—Case 3. An x-ray of a 32-year-old man after a left extrapleural pneumonectomy for a destroyed left lung (no oxygen uptake or ventilation on the left side by bronchospirometry) and a tuberculous empyema in an extrapleural pneumothorax space performed eight years earlier.

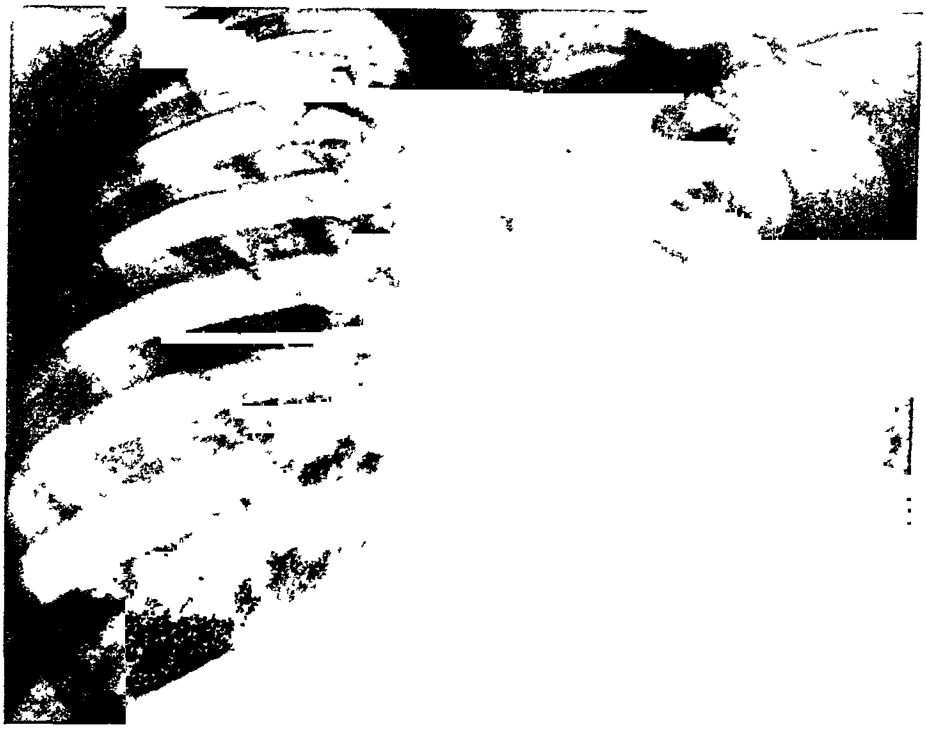

Fig. 10.—Case 4. An x-ray of a 23-year-old man one year after a left pneumonectomy for a destroyed left lung behind a stenosis of the main bronchus. Ribs 2 and 4 were bent down and fixed across the mediastinum. The procedure has resulted in considerable herniation.

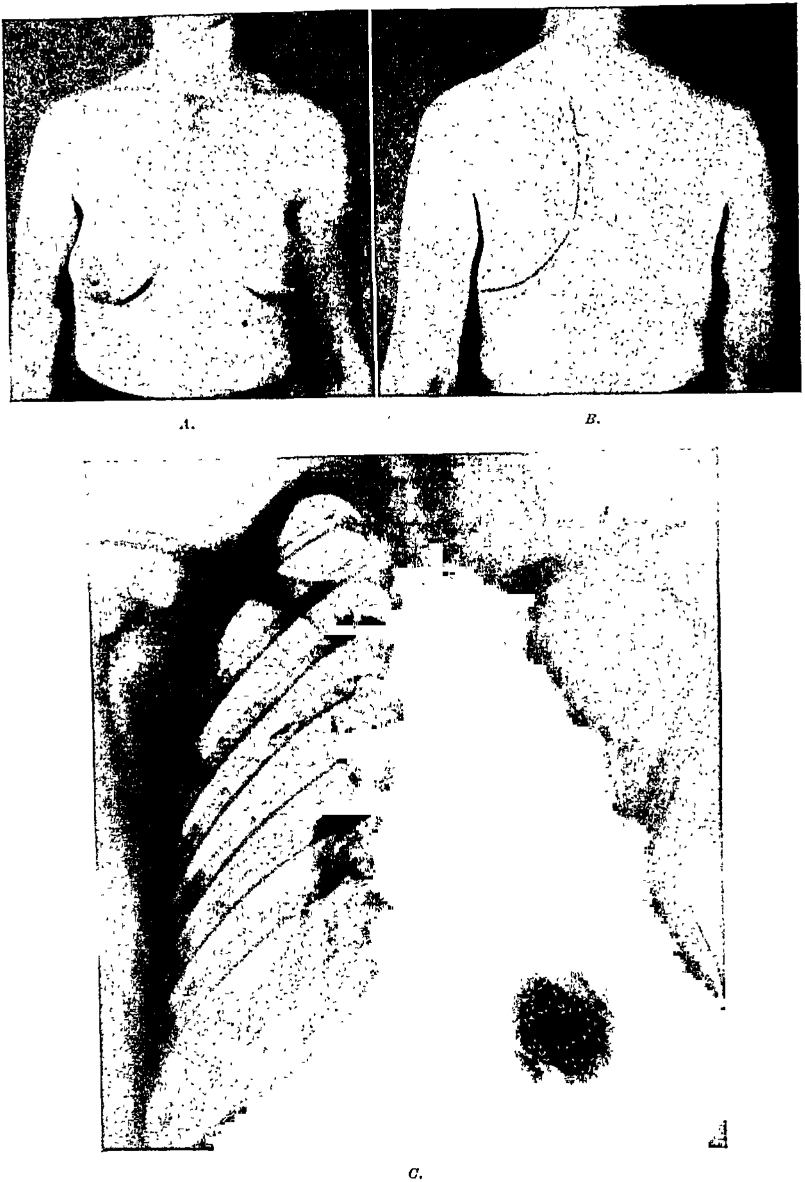

Fig. 11.—*A*, *B*, and *C*. Case 5. The cosmetic result in a 30-year-old woman one year after an extrapleural pneumonectomy with osteoplastic wall thoracoplasty. The first rib was left in place in this case. The patient is working full time. [illegible] maxi[illegible] breathing capacity is 58 L. per minute, the residual capacity 24 per cent of [illegible]e is only a slight scoliosis. The mediastinum is kept in the midline.

Case 3.—A 32-year-old man developed a tuberculous cavity in the left upper lobe in 1946. An extrapleural pneumothorax was performed in 1947 but was complicated with a tuberculous empyema. There were, furthermore, remaining cavities in both upper and lower lobes on the left side and in the upper lobe on the right side. The maximal breathing capacity was 50 L. per minute with 100 per cent of the oxygen uptake and ventilation on the right side. An extrapleural pneumonectomy with osteoplastic wall thoracoplasty was performed on the left side (Fig. 9).

Case 4.—A 23-year-old man had a left pleurisy in 1945. Since 1951, a left main bronchial stenosis had developed with gradual destruction of the left lung. The resting arterial oxygen tension was 87. A left pneumonectomy was performed and at the same time ribs 2 and 4 were bent down across the mediastinum and the heart and fixed to the transverse processes of the sixth and ninth vertebrae. One year after operation, some herniation had developed (Fig. 10). The residual capacity, however, remained 22 L. per minute which was the same as before the operation.

DISCUSSION

A one-stage procedure has been devised to obliterate the space after pneumonectomies. By the immediate obliteration of this space, the danger of infection is diminished which is essential especially in cases with extrapleural resections for empyema.

A stable chest wall against the mediastinum is obtained. This new chest wall will prevent paradoxical movements in the immediate postoperative period. By earlier techniques for total thoracoplasty, it has only been possible to diminish the hemithorax by two-thirds of its original size. Thus, some overexpansion of the remaining lung and a displacement of the mediastinum to the operated side will nearly always follow in spite of the extensive rib resection.

By the new osteoplastic thoracoplasty, the ribs are placed across the mediastinum and fixed to the vertebral body. Thereby, a central position of the mediastinum may be maintained and herniation with overdistention of the remaining lung prevented.

SUMMARY

An osteoplastic wall thoracoplasty has been devised for obliteration of the space after pneumonectomies for tuberculosis in a one-stage procedure.

Paradoxical movements are prevented and the mediastinum maintained in a central position. The procedure has been performed on 20 patients with three deaths.

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HYPOPLASIA OF THE LUNG IN CONGENITAL DIAPHRAGMATIC HERNIA

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HERNIAS of the lumbocostal triangle of Bochdalek are the most common type of congenital diaphragmatic hernia encountered in the newborn but are extremely rare in adults.[1, 2] This is due to the fact that most of the infants affected by this condition die in the first days after birth owing to cardiorespiratory failure. Among 50 newborn infants with this type of hernia, 12 survived less than one hour, 9 less than twenty-four hours, and 2 less than thirty-six hours.[3] A review of 210 cases of congenital diaphragmatic hernia revealed that, without operative treatment, 75 per cent of the infants died before the end of the first month of life.[4] Prompt surgical intervention in the postnatal period when the viscera are not yet distended by air is therefore strongly advocated.

However, in some cases the presence of abdominal viscera in the thorax is well tolerated, apparently, and the infant may grow to childhood and even to adult life. The diaphragmatic hernia, in such cases, may be discovered only on routine x-ray examination, as was the case in the patient whose history follows.

CASE REPORT

P. N., a twelve-year-old boy, was admitted to the Department of Chest Diseases of the Hadassah-University Hospital, Jerusalem, Israel, on June 30, 1952. His previous history was uneventful. In October, 1951, on mass radiography, the lower two-thirds of the left thorax was found to be opaque (Fig. 1). This finding was interpreted as pleural effusion and when a pleural tap was attempted, the boy fainted and was admitted to a hospital where he stayed for one week. In June, 1952, pain developed in the left chest accompanied by fever and the boy was admitted to our hospital. On routine examination, the patient appeared normally developed for his age but was poorly nourished. His weight was 28 kilograms and his height 133 cm. Mild scoliosis was noted. The temperature was 36.9° C. to 37.5° C., the pulse 80, the blood pressure 100/65, respirations were 20 per minute, and the erythrocyte sedimentation rate was 60/75. Two weeks later the temperature and erythrocyte sedimentation rate became normal. Hyperresonance to percussion over the left chest was found with absence of normal vesicular breathing and vocal fremitus. The heart sounds were normal. The liver and spleen were not palpable. A blood count showed 4.13 million red blood cells, 12 Gm. hemoglobin, 10,200 white blood cells with 55 per cent neutrophils, 39 per cent lymphocytes, 5 per cent eosinophils, and 1 per cent monocytes. Urine analysis was normal. Serologic tests for syphilis were negative. The Mantoux test in 1/1,000 was positive. Gastric content smears and cultures were negative for acid-fast bacilli. An electrocardiogram revealed right axis deviation. The pulmonary function studies showed the vital capacity to be 40 per cent of the predicted value and the maximal breathing

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Table I. Ventilatory Function Tests

	AGE	HEIGHT (CM.)	WEIGHT (KG.)	VITAL CAPACITY OBSERVED (ML.)	PER CENT OF PREDICTED VITAL CAPACITY	TIDAL AIR (ML.)	INSPIRATORY RESERVE (ML.)	EXPIRATORY RESERVE (ML.)	MAXIMUM BREATHING CAPACITY OBSERVED (L./MIN.)	PER CENT OF PREDICTED MAXIMUM BREATHING CAPACITY	AIR FLOW VELOCITY INDEX
Before operation	12	144	30	1030	40	200	531	350	43	47	1.17
After operation (2 months)	12.5	146	31	775	31	200	400	175	30	31	1
After operation (2½ years)	15	166	46	2080	48	673	717	690	71.2	60	1.25

capacity 47 per cent of the predicted value (Table I). Bronchoscopy showed a marked narrowing of the openings of left main and particularly of the left upper and lower lobe bronchi. With bronchoscope No. 6, it was impossible to enter farther than the left-main bronchus.

Chest x-ray revealed the presence of air in the upper part of the opacity in the left thorax and diaphragmatic hernia was therefore suspected (Fig. 1). A barium meal and a barium enema showed that all the small intestine, cecum, and colon were in the left

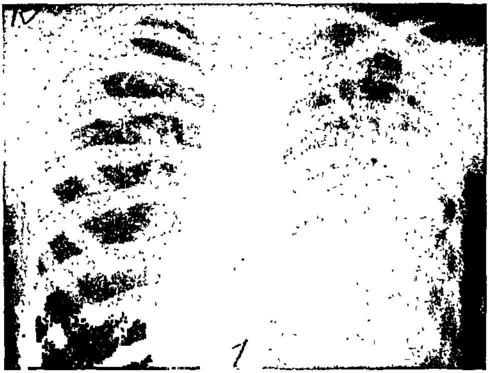

Fig. 1.—Chest x-ray showing an opacity in the lower part of the left thorax due to herniated intestines.

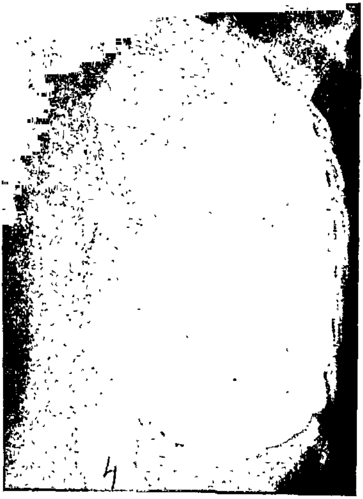

Fig. 2.—X-ray studies after a barium enema to demonstrate the left thorax filled with bowels.

thorax (Figs. 2 and 3), and that the communication between the thorax and abdomen was in the posterior part of the thorax. The left diaphragm was not seen on x-ray and no movements were visible on fluoroscopy. However, a diagnostic pneumoperitoneum (Fig. 4) revealed diaphragmatic movements on screening. Bronchography showed very poor filling of the left bronchial tree, characteristic of hypoplastic lung (Fig. 5). On pyelography, no pathologic changes were found.

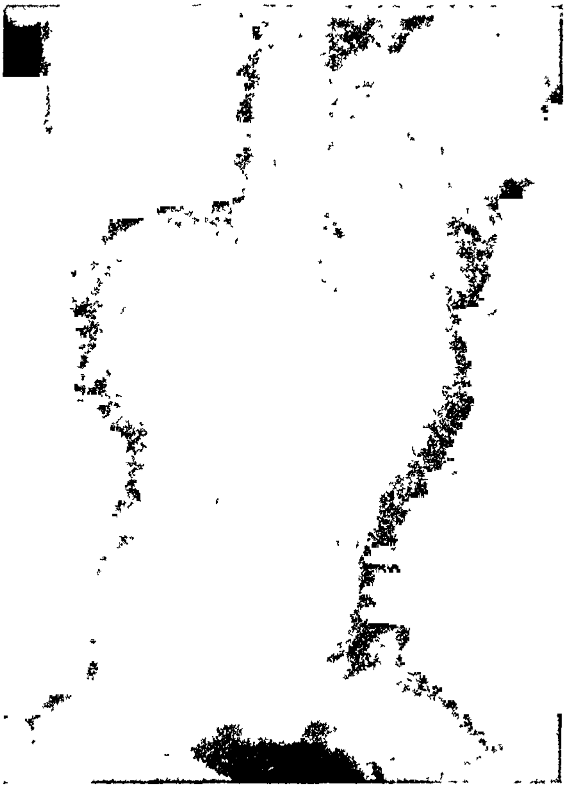

Fig. 3.—Roentgenogram after barium meal showing the stomach low in the abdomen and small bowel loops in the left chest.

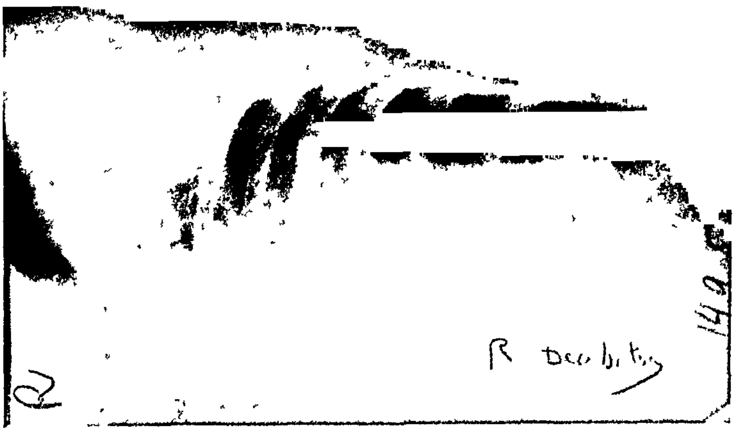

Fig. 4.—Roentgenogram taken after induction of pneumoperitoneum demonstrating pneumothorax on the left side.

The clinical diagnosis was congenital diaphragmatic hernia through the lumbocostal triangle with hypoplasia of the left lung.

Operation.—(H. M.) Under endotracheal anesthesia, the left thorax was opened through the seventh intercostal space. The pleural cavity was almost completely filled with intestinal loops without parietal peritoneal coverings. The left lung was the size of that seen in a 2-year old child but showed normal development of both lobes. The lower lobe was atelectatic but could be inflated by positive pressure. The defect in the diaphragm was found in its posterolateral part and measured 10 cm. by 6 cm. Through this defect nearly all of the small bowel (from the upper jejunum to the lower ileum) and the ascending, transverse, and descending colons protruded from the abdominal into the chest cavity. There were many adhesions between the intestines and the margins of the diaphragmatic defect, and these had to be severed before the bowel could be replaced into the abdomen. The defect in the diaphragm was repaired by two rows of mattress sutures. The left phrenic nerve was crushed. The hypoplastic lung was inflated by the anesthetist but filled only one-third to one half of the thoracic cavity which was then closed with underwater-seal drainage.

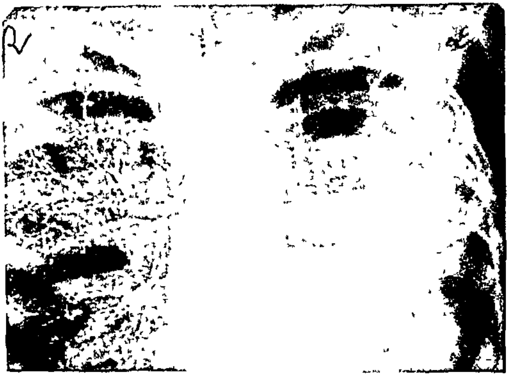

FIG. 5.—Bronchogram demonstrating poor filling of the left bronchial tree.

The postoperative course was somewhat difficult during the first week owing to marked abdominal distention which required the use of Wangensteen suction. Thereafter, however, normal bowel function was resumed and the boy became free of abdominal discomfort.

After operation, the left lung expanded a good deal and vesicular breathing, although weaker than on the right side, became audible, particularly in the upper part of the hemithorax. This expansion was confirmed by a second bronchography (Fig. 6) which showed good filling of the upper and lingular bronchi. However, the lower bronchus still filled poorly. The hypoplastic lung did not fill the entire hemithorax and six weeks after operation a pleural effusion appeared. The pleural dead space, created after the intestines were replaced in the abdomen, was thereafter filled by fibrinous organization of the pleural fluid, elevation of the diaphragm, and mediastinal shifting. Two months after operation, respiratory function tests showed a decrease in vital capacity of 9 per cent and of the maximum breathing capacity of 16 per cent, the result of pleural involvement and phrenicolysis. Two and one half years after operation, the boy was well and x-ray showed good expansion of the lung (Fig. 7). Pleural thickening was moderate, vital capacity was 48 per cent, and maximum breathing capacity 60 per cent of the predicted values (Table I).

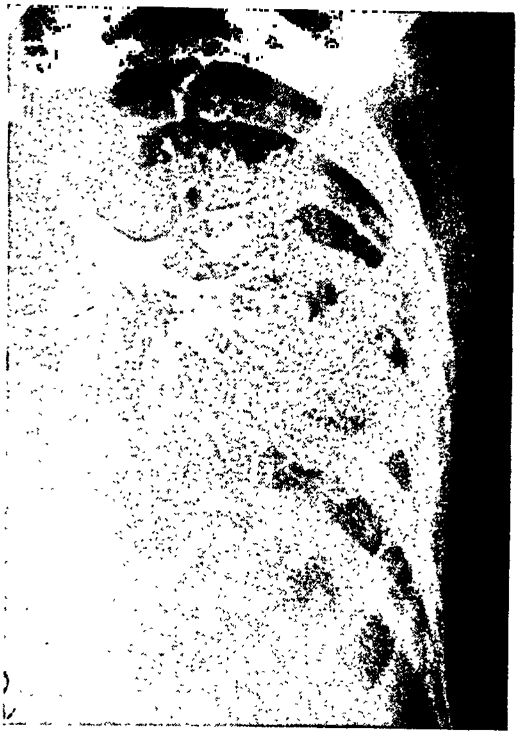

Fig. 6.—Bronchogram taken eight months after operation showing a good filling of the bronchi of the left upper lobe and poor filling of the lower lobe.

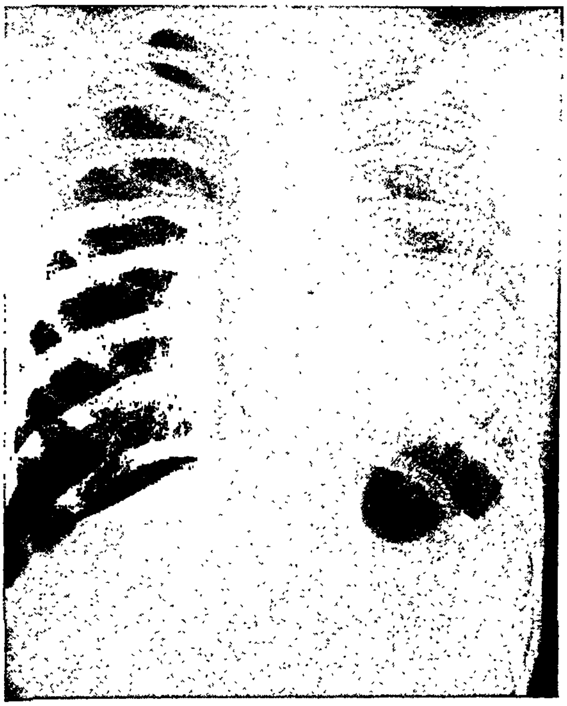

Fig. 7.—Roentgenogram taken two and one-half years after operation showing good re-expansion of the left lung.

DISCUSSION

Although the subject of congenital diaphragmatic hernia is extensively dealt with in surgical literature and the intestinal complications are well known, little attention has been paid to the state of the lung in this condition In hernia of the foramen of Bochdalek, the lung and mediastinum are usually compressed by the large mass of abdominal viscera which has entered the thorax and only few infants escape early death If complications from congenital diaphragmatic hernia arise during childhood and adolescent life, they are due mostly to disturbed bowel function such as intestinal obstruction, strangulation, etc, and this may account for the fact that little mention is made of the fate of the lung in this condition In addition, many surgeons employ and advocate exclusively the abdominal approach for the repair of these diaphragmatic hernias, particularly on the left side They believe that reduction of the viscera is easier by traction from below than by pulsion from above[6] It is evident that the use of the abdominal approach precludes proper visualization and palpation of the lung and, thus, pulmonary pathology will escape detection and adequate treatment Other authors recommend the thoracic approach because of the better exposure obtained, the possibility of more rapid expansion of the lung, and the much smoother convalescence[7-9] We employ the thoracotomy approach on hernia of foramen of Bochdalek and find it superior to the abdominal approach We feel that the lung should be carefully examined in every case and that inflation should be instituted as soon as the eventrated viscera have been returned to the abdominal cavity

We have found only two instances in the literature where the state of the lung in congenital diaphragmatic hernia is mentioned Bonilla Naar and co workers[5] report on a 10 year old boy who complained of dyspnea and in whom diaphragmatic hernia was diagnosed At operation, a completely atelectatic left lung was found The upper lobe was inflated but the lower lobe remained airless and functionless These authors used the lower lobe to plug the large defect in the posterolateral part of the diaphragm Fisher[10] reported on a 12 year old girl with diaphragmatic hernia in whom the bronchogram showed a filling defect in the left lower lobe During closure of the diaphragmatic defect, an atelectatic and hypoplastic lower lobe was found

In our case, the hypoplastic lung was inflated and its function partially restored The postoperative x ray studies and ventilatory function tests showed a marked improvement in pulmonary function in spite of phrenic interruption and pleural fibrosis

SUMMARY

A case of congenital diaphragmatic hernia through the foramen of Bochdalek with hypoplasia of the lung is reported The problem of the state of the lung in cases of diaphragmatic hernia is discussed and the thoracic approach for the repair of these hernias is recommended

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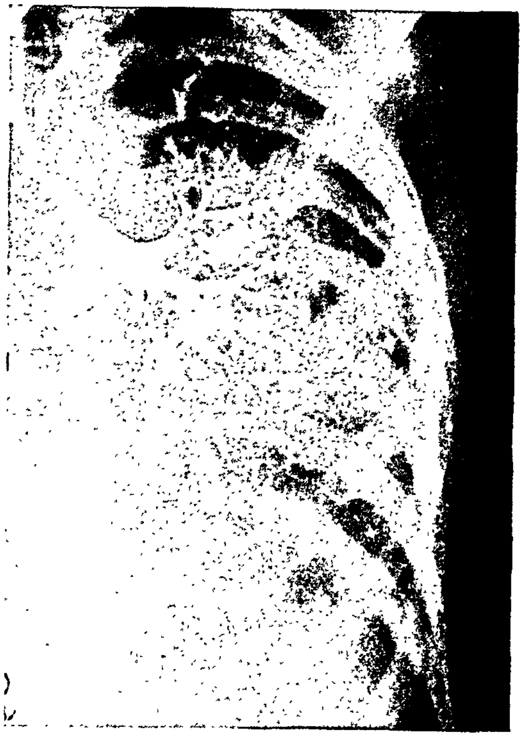

Fig. 6.—Bronchogram taken eight months after operation showing a good filling of the bronchi of the left upper lobe and poor filling of the lower lobe.

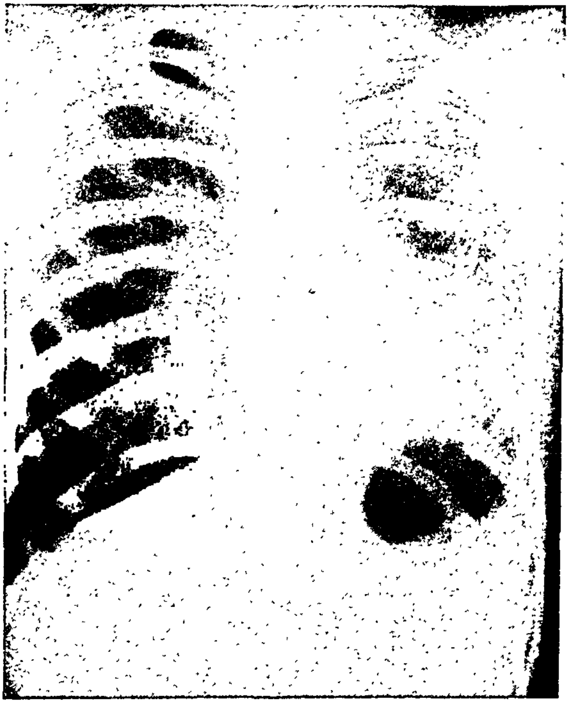

Fig. 7.—Roentgenogram taken two and one-half years after operation showing good re-expansion of the left lung.

DISCUSSION

Although the subject of congenital diaphragmatic hernia is extensively dealt with in surgical literature and the intestinal complications are well known, little attention has been paid to the state of the lung in this condition. In hernia of the foramen of Bochdalek, the lung and mediastinum are usually compressed by the large mass of abdominal viscera which has entered the thorax and only few infants escape early death. If complications from congenital diaphragmatic hernia arise during childhood and adolescent life, they are due mostly to disturbed bowel function such as intestinal obstruction, strangulation, etc., and this may account for the fact that little mention is made of the fate of the lung in this condition. In addition, many surgeons employ and advocate exclusively the abdominal approach for the repair of these diaphragmatic hernias, particularly on the left side. They believe that reduction of the viscera is easier by traction from below than by pulsion from above.[6] It is evident that the use of the abdominal approach precludes proper visualization and palpation of the lung and, thus, pulmonary pathology will escape detection and adequate treatment. Other authors recommend the thoracic approach because of the better exposure obtained, the possibility of more rapid expansion of the lung, and the much smoother convalescence.[7-9] We employ the thoracotomy approach on hernia of foramen of Bochdalek and find it superior to the abdominal approach. We feel that the lung should be carefully examined in every case and that inflation should be instituted as soon as the eventrated viscera have been returned to the abdominal cavity.

We have found only two instances in the literature where the state of the lung in congenital diaphragmatic hernia is mentioned. Bonilla-Naar and co-workers[5] report on a 10-year-old boy who complained of dyspnea and in whom diaphragmatic hernia was diagnosed. At operation, a completely atelectatic left lung was found. The upper lobe was inflated but the lower lobe remained airless and functionless. These authors used the lower lobe to plug the large defect in the posterolateral part of the diaphragm. Fisher[10] reported on a 12-year-old girl with diaphragmatic hernia in whom the bronchogram showed a filling defect in the left lower lobe. During closure of the diaphragmatic defect, an atelectatic and hypoplastic lower lobe was found.

In our case, the hypoplastic lung was inflated and its function partially restored. The postoperative x-ray studies and ventilatory function tests showed a marked improvement in pulmonary function in spite of phrenic interruption and pleural fibrosis.

SUMMARY

A case of congenital diaphragmatic hernia through the foramen of Bochdalek with hypoplasia of the lung is reported. The problem of the state of the lung in cases of diaphragmatic hernia is discussed and the thoracic approach for the repair of these hernias is recommended.

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PANCOAST TUMOR

Five-Year Survival Without Recurrence or Metastases Following Radical Resection and Postoperative Irradiation

William M. Chardack, M.D., Buffalo, N. Y., and James D. MacCallum, M.D., Warsaw, N. Y.

PAIN, paralysis, and atrophy within the lower brachial plexus territory, Horner's syndrome, and a density in the pulmonary apex with erosion of adjacent ribs are the salient features of the Pancoast syndrome. Although similar isolated cases had been described prior to Pancoast's original publication in 1924,[12] he was the first to draw wide attention to the entity now bearing his name.

Three typical cases formed the basis of his report. A biopsy had been taken in 2 of the 3 cases and had revealed a malignant tumor; death had occurred rapidly in all 3 from what he termed a "comparatively trivial growth without detectable metastases."

Pancoast thought he was dealing with a new and specific clinical entity, a carcinoma arising from epithelial rests of the fifth branchial pouch.

In 1932, addressing the Radiological Section of the American Medical Association, he[13] reported 4 additional cases, suggested that the lesion be called "superior sulcus tumor," and restated his belief in its branchial origin although he prudently added: "It is possible that this designation may be changed again with a better knowledge of the histopathology of the growth."

Herbut and Watson[8] reviewed the literature on this subject in 1946, collected 151 cases, concluded that a variety of malignant tumors can indeed produce this syndrome and proposed the comprehensive designation of "tumor of the thoracic inlet producing the Pancoast syndrome." In 1953, in our preliminary report on this case,[3] we reviewed all contributions to this subject published subsequently to Herbut and Watson's study and this perusal of the world literature has been brought up to date. Mention will be made in the text only of those publications dealing with new and pertinent facts. For a detailed description of the syndrome, the reader is referred to Herbut and Watson's article. We shall limit ourselves to a summary of the present concept of this lesion and a follow-up report on a five-year survival and possible cure following removal of such a tumor.

The anatomic location of this lesion within the confines of the thoracic inlet determines its clinical features; the latter are the result of compression and invasion of the brachial plexus, sympathetic chain, and the surround-

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ing ribs. Depending upon size, stage of development, and invasive qualities of the lesion, the syndrome may be incomplete or additional features may appear as further neighboring structures, vascular or other, become engulfed by the tumor.

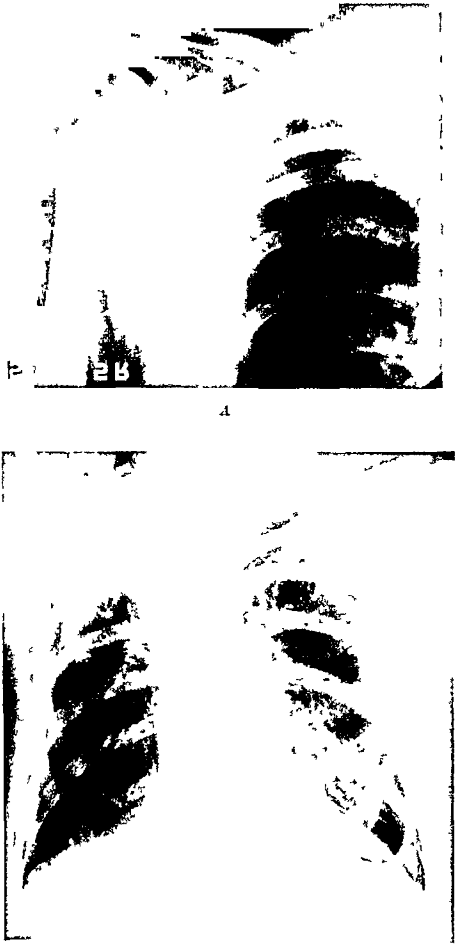
With regard to the origin of the lesion, Pancoast's concept of a specific branchiogenic epithelioma was discarded as subsequent observations showed that a wide variety of malignant tumors may produce a Pancoast syndrome, either by direct extension into or by metastases located in the thoracic inlet. However, the vast majority of such cases are due to a bronchogenic carcinoma arising in the extreme pulmonary apex.

There has been unanimity of opinion in the past on the rapidly fatal course of these tumors. Indeed, it is widely held that the lesion is an incurable one and that the presence of a Pancoast syndrome signifies inoperability and prohibits surgical exploration.

Past experience, it is true, had justified the pessimism expressed with regard to the prognosis of a Pancoast syndrome. Herbut and Watson[8] stated in 1946, "to date the disease has been 100 per cent fatal. Roentgen therapy so far appears of no avail." They suggested, however, that in the future an operable case might be detected and urged thorough investigation, even by surgical exploration if necessary, of any persistent neuritic pain of the upper limb. Walker,[14] in 1946, wrote "death has occurred in all cases recognized up to the present time. In no instance has it been possible to remove the tumor surgically and irradiation has not been of palliative benefit."

A review of the literature up to the date of this publication has failed to yield any authenicated cases of a successful resection of such a tumor with the exception of the 1 reported by the authors in 1953. Actually only seven explorations of these lesions had by then been recorded, and in no instance had the tumor been resected. More recently, Becker,[1] in 1953, recorded an exploratory thoracotomy but the tumor was nonresectable. Hamm,[7] in 1952, stated that only operative treatment might improve the prognosis and he quoted Frey who is said to have reported a successful surgical resection of such a tumor to the Congress of Internal Medicine in 1951. Frey's[5] address, however, does not contain any reference to a Pancoast tumor. The presentation dealt with recent advances in thoracic surgery and made mention only of a case of a sarcoma of the lung in the dependent portion of the left upper lobe with invasion of the chest wall in this lower area.

While the experience with x-ray treatment of this lesion had also been uniformly disappointing in the past and the majority of reports indicated that it had been of no avail, there have been some recent exceptions to this view. Hamm[7] records a case of undifferentiated epithelioma producing a Pancoast tumor and proved by biopsy. He states that delivery of 2,800 r led to a surprise remission which had lasted for more than one year at the time of his publication and the patient was then still under observation. Haas and associates,[6] in 1954, stated that they had relatively better success with Pancoast type lesions than with other bronchogenic neoplasms. They had treated 4 patients with such tumors and, of these, 1 was living and well thirty-four months after irradiation. There had been regression of the mass as well as recalcification of the rib after delivery of 11,063 r by betatron therapy. No details are given with regard to histologic proof of the nature of the lesion. Miceli,[11] in 1952, stated that he had obtained some temporary relief by x-ray therapy.



A

B

Fig 1.—X ray photograph of the right shoulder and apex of the lung at time of admission (*A*) and five years after operation (*B*). (*A*, From Chardack and MacCallum, J Thoracic Surg, April, 1953.)

Malcolmson,[9] in 1947, reported a case in which irradiation had produced the regression of a large mass in the left upper lobe. The patient had been followed for eighteen years and the tentative diagnosis suggested by the Mayo Clinic was one of lymphoblastoma. Formal proof by biopsy had not been obtained.

It may then be stated that only a very few patients with Pancoast tumors have had exploratory operations, that we know of no other instance of a successful resection, and that in the opinion of the majority of writers, x-ray therapy has been of no avail in proved carcinoma with the exception of the previously quoted instances in none of which, however, has it produced an authenticated cure in a proved malignant tumor.

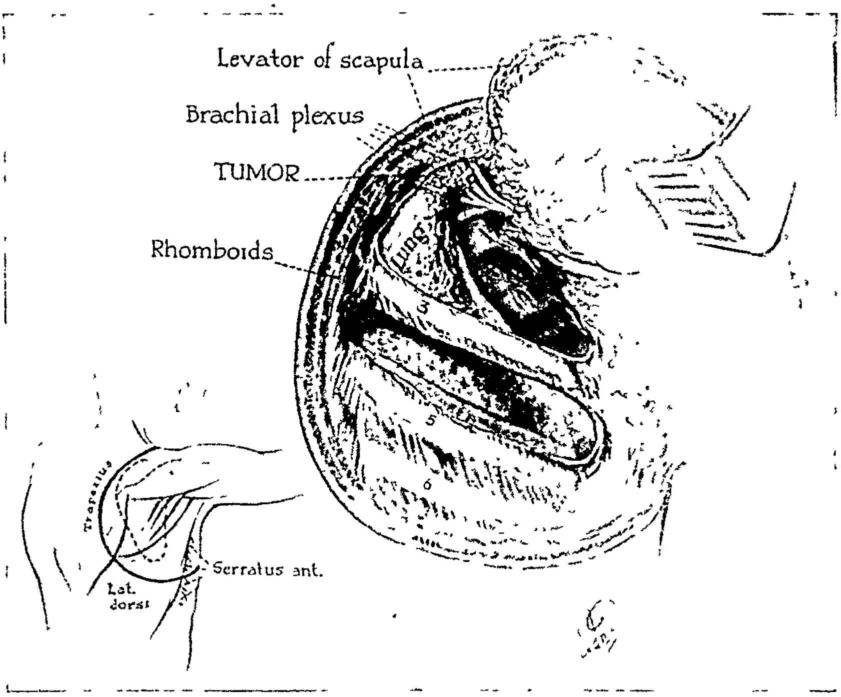
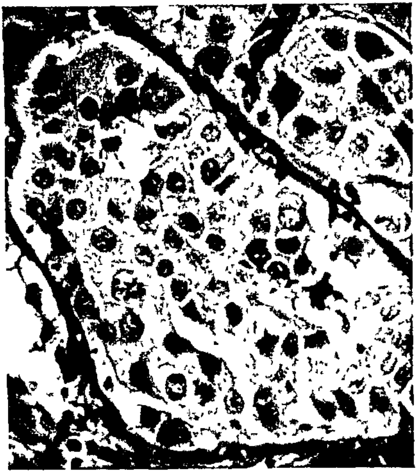


Fig. 2.—Artist's conception of the operative field and the line of incision. (From J. Thoracic Surg., April, 1953.)

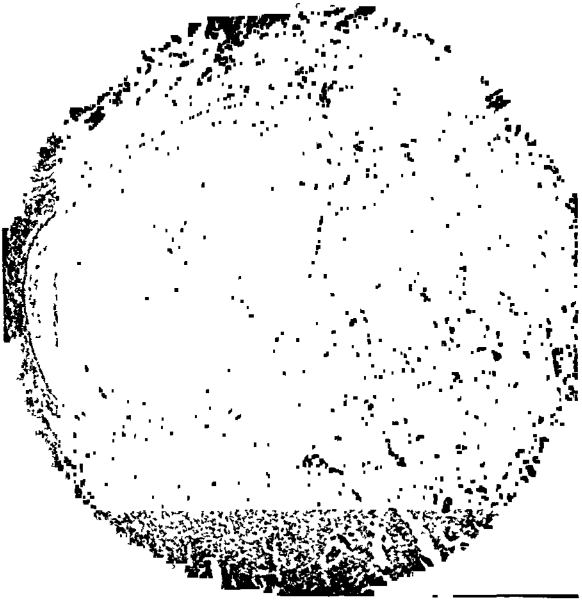
CASE REPORT

H. R., a 54-year-old white man, was admitted to the Veterans Administration Hospital in Buffalo, N. Y., on April 19, 1950, complaining of excruciating and intractable pain around the shoulder which radiated into the ulnar territory. A paralysis of ulnar distribution was present. There were ptosis and myosis of the right eye and an x-ray of the chest showed a small density in the right apex (Fig. 1, *A*). Additional laboratory findings were noncontributory. The clinical diagnosis was apical bronchiogenic carcinoma with Pancoast syndrome and surgical intervention was carried out on May 15, 1950.

Operative Procedure.—For a detailed description of technique, the reader is referred to our original report in 1953.[3] Suffice it to say here that through a long periscapular incision, the tumor was removed in toto by a right upper lobectomy and en bloc excision of the involved ribs, pleura, endothoracic fascia, and spinal nerve roots at the level of the first and the eighth thoracic vertebrae, and the seventh cervical vertebra. Fig. 2 shows surgical exposure and the tumor extending through the apex of the pleural dome into the brachial plexus and it was felt that by sacrificing the invaded roots, there was a reasonable chance to remove the



A.

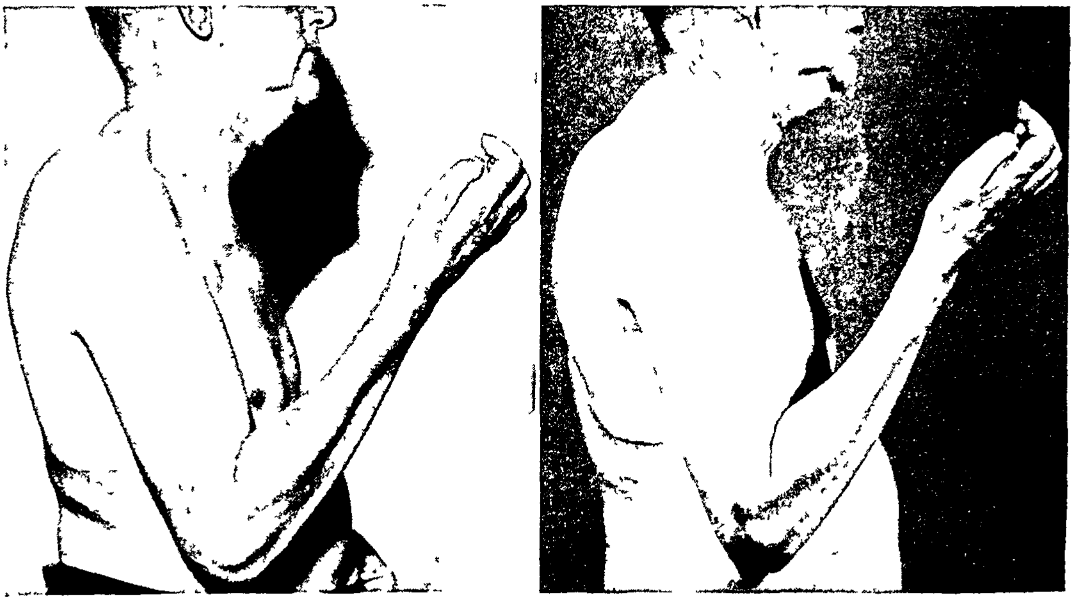


B.

Fig. 3.—High power microphotograph of the tumor tissue from the intrapulmonary component (*A*), and microphotograph showing invasion of a brachial plexus nerve (*B*). (From J. Thoracic Surg., April, 1953.)

entire growth. Pathologic examination of the specimen revealed a highly undifferentiated squamous cell carcinoma, clearly originating in and traceable to one of the minor branches of the right upper-lobe bronchus. Invasion by the tumor of the perineural spaces of the brachial plexus cord is demonstrated on the photomicrograph in Fig. 3.

Postoperative Course and Follow up.—The postoperative course was uneventful. Neurologic examination revealed the expected deficit due to removal of roots at the level of the seventh and eighth cervical vertebrae and the first thoracic vertebra but the postoperative changes were hardly more extensive than the damage from tumor involvement observed preoperatively.



A. *B.*

Fig. 4.—Photographs of the patient in 1952 (*A*), and in April, 1955 (*B*). (*A*, From J. Thoracic Surg., April, 1953)

Although recorded experience with radiotherapy had been disappointing, it was felt that, in this instance, with all gross carcinoma removed and the overlying bony structures resected, the possibility of delivering a cancerocidal dose to any potentially remaining microscopic foci of an undifferentiated tumor was greatly enhanced. Accordingly, three weeks after surgery, radiation therapy was begun and 6,528 r (half value layer copper 25 mm.) were delivered over fifty-four days to the general tumor area.*

The patient was followed at approximately three-month intervals and his present status five years after the operation is as follows: he weighs approximately one hundred seventy pounds, ten pounds more than before the operation and his weight has been stable since 1951. His general condition is excellent. There is no evidence of recurrence or metastasis and there has been no increase in the extent of his paralysis. Trophic changes of the tissues of the hand are marked. He complains of an occasional mild, burning sensation in the right hand but grades this as negligible compared to the pain sensation before the operation. This slight discomfort is entirely acceptable to the patient and he does not require medication. There is no interference with sleep. He has adjusted to the disability resulting from the paralysis and feels that the limb is still partially useful to him. He was last examined in April, 1955.

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*Therapy was carried out by Dr. Walter T Murphy, Roswell Park Memorial Institute, Buffalo, N. Y., Consultant Radiotherapist to the VA Hospital, Buffalo, N. Y.

DISCUSSION

Since, in the view of many surgeons, it is widely believed that the presence of a Pancoast tumor signifies inoperability and incurability and constitutes a contraindication to exploration, we feel that, in the light of our experience, this concept deserves revision It has been shown that a Pancoast tumor, or more specifically, that a bronchogenic carcinoma with Pancoast syndrome has been successfully treated by radical resection of all involved structures, followed by radiotherapy Several aspects deserve further discussion

Of particular interest is the question as to whether a Pancoast syndrome is invariably and exclusively due to a malignant tumor or whether a benign tumor or even a nonneoplastic lesion may on occasion be responsible for a similar symptom complex Both questions can be categorically answered in the positive since our review of the literature, in 1953, yielded 6 such authenticated cases In 1 instance, a neuroma was responsible for the syndrome, tuberculosis accounted for 3 and a Hand Schuller Christian disease and a nonspecific pneumonitis of the left upper lobe for the remaining 2 cases Since then, similar experiences have come to light D'Eschougues and Houel,[4] in 1954, reported a Pancoast syndrome due to the presence of a fist sized hydatid cyst removed by thoracotomy Bellion,[2] in 1953, and Marques and associates,[10] in 1952, reported the presence of a Pancoast syndrome due to Hodgkin's disease proved by biopsy and treated radiologically The importance of these observations is considerable since they indicate that it is the location and not the nature of the expanding lesion that is responsible for the genesis of the syndrome Far from contraindicating surgical exploration, the presence of a Pancoast syndrome imperatively dictates intervention since an entirely benign lesion and one not necessarily amenable to radiotherapy may be its cause

The question may be raised as to the part played by the radiation therapy in the cure of our case In past experience, radiotherapy alone has not been capable of achieving a cure of a Pancoast syndrome due to a proved malignant tumor Radical surgical removal followed by intensive radiotherapy may well be more potent than either method alone, when one is dealing with a tumor showing invasion of preformed and lymphatic spaces such as was the case here It seems logical to assume that while all grossly recognizable tumor tissue with an apparently adequate margin of normal tissue is removed by radical surgery, microscopic foci may remain beyond the field of excision and it is conceivable that a cancerocidal dose delivered to a relatively wide field may be of value, especially when overlying soft tissues and bony structures have been removed In any event, the combination of extensive removal followed by radiotherapy was capable of dealing with a lesion heretofore considered to be always rapidly fatal

In addition to the now proved possibility of achieving at least a five year survival without demonstrable recurrence or metastases, surgical removal is capable of producing important palliation in a disease notorious for the unbearable pain and discomfort it produces Even if not entirely removable, partial ablation of a tumor in this particular location is justifiable since it can produce impressive pain relief Since our original communication in 1953,[3] we

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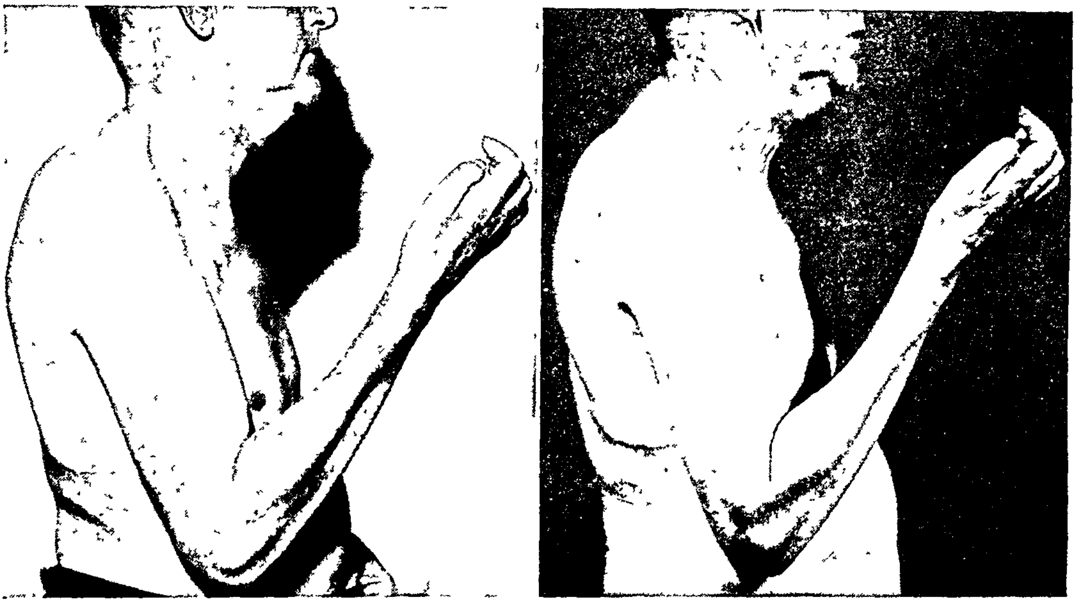
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SUMMARY AND CONCLUSIONS

1. A follow-up report on a case of Pancoast syndrome due to bronchogenic carcinoma is presented. This lesion, generally thought to be inoperable, has been successfully treated by en bloc resection of the tumor and adjacent involved structures, followed by irradiation. The patient is now well and free of recurrence and metastases five years after the procedure.

2. The prevailing concept that the Pancoast syndrome signifies inoperability and incurability should be reconsidered and any unexplained persistent neuritic pain in the upper limb should be thoroughly investigated with the possibility of a Pancoast syndrome-producing lesion in mind. Earliest surgical exploration is justified because: (a) the lesion may be benign and not necessarily radiosensitive, (b) if it is malignant, it may be resectable and possibly be curable, and (c) if it is not resectable, important palliative benefits may be achieved by decompression in an otherwise hopeless and extremely distressing type of tumor.

Addendum: The patient was seen last at this hospital in February, 1955. He was well. On March 13, 1956, he died after a brief illness, five years and ten months after his operation. A post-mortem examination was granted and no evidence of recurrence or metastases was found. The thoracic organs as well as the contents of the right supraclavicular triangle were dissected grossly and studied microscopically by Dr. Kornel L. Terplan, Professor of Pathology, University of Buffalo School of Medicine. The cause of death was bronchopneumonia, mild cor pulmonale, and myocardial failure. There was no evidence of tumor.

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THE PROBLEM OF CARCINOMA IN ACHALASIA

STANLEY E. POTTER, M.D., JOHN A. RASMUSSEN, M.D., AND
R. RUSSELL BEST, M.D.
OMAHA, NEB.

THOSE who explore for the first time the literature on this interesting subject are met immediately by a number of paradoxes, most of which are difficult to resolve.

The reported number of cases is remarkably small, but more remarkable is the fact that certain cases have been excluded from a review of the literature, "where it has not been possible, owing to wartime dispersal of medical libraries, to study the original article."[1] Other cases have not been included in tabulated reviews because of "insufficient clinical details."[1]

We feel that to perpetuate the figure of 15 or 16[2, 3] reported cases accomplishes nothing, for reported cases are no index of occurrence particularly when their very numbers are suspect.

Many more imponderables arise. Gottstein[5] reported the incidence of carcinoma in achalasia in his series at 10 per cent, Rake[6] reported an incidence of 20 per cent. Poles apart, statistically, is Vinson[7] who makes no mention of carcinoma in reporting 683 cases of achalasia.

When one approaches the problem from the aspect of esophageal carcinoma, essential agreement with infrequency is found. In an experience with over 600 cases of carcinoma of the esophagus, Puestow had none associated with achalasia.[8]

Attempts to resolve these opposites have the appeal of rationalizing that the European incidence is higher, or that our identification of this disease association is careless or not recorded. We have no proof of the former but we believe the latter possible, in our own geographic area at least.

Much stress has been placed upon repeated esophagoscopic examinations in achalasia to detect the development of carcinomatous change. This is indeed worthy. We feel also that the periodic esophagogram done by an experienced radiologist in a well prepared esophagus will be useful and may provide the necessary periodic follow up for more patients. In reviewing the assembled cases, we were impressed by the high incidence of positive radiologic diagnoses of tumor, if the possibility were kept in mind.

Stimulated by a recent case of our own, we have reviewed all detailed cases in the English literature in an effort to find an early clue to this formidable complication of achalasia (Table I).

From the University of Nebraska College of Medicine, Omaha, Neb.

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TABI

	SEX	AGE	DURATION OF ACHALASIA	ASPIRATION PNEUMONIA	BLEEDING	ANEMIA	WEIGHT LOSS	CHANGE IN HISTORY
	F	47	10 years	None	Not stated	Yes	"Several pounds"	Increase in severity an loss of intermittenc of dysphagia
lum and er[10] Case III	F	47	10 years	Questionable	Yes	Yes	Yes	Not stated
lum and er[10] Case IV	M	46	18 years	None	Not stated	Not stated	Yes	Cough and weight los
and nedy[7]	M	34	8 years	None	Not stated	Not stated	Yes	Progressive dysphagia
[4]	M	47	10 years	None	Not stated	No	Yes	Progressive dysphagia
nd Sicher[1]	M	65	20 years	None	Yes; vomitus	Not stated	Emaciation	Increasing dysphagia; hematemesis
and Lam[2]	M	42	10 years	None	Yes; vomitus	Not stated	30 pounds	Hematemesis; return o dysphagia on conservative management
	M	56	24 years	Yes	Not stated	Yes	30 pounds	Chills, fever, productiv cough
, Rasmussen Best	M	51	4-5 years	Yes	No	No	20 pounds	Progressive dysphagia

The sex incidence, as with carcinoma of the esophagus alone, is predominantly male. The age incidence is that of carcinoma in general.

It would appear that carcinoma favors long-standing achalasia; the average duration of proved or suspected esophageal dilation being twelve years. This fact, along with the low incidence of tumor, is difficult to reconcile with the belief that chronic irritation is important in the development of epidermoid carcinoma.

Aspiration pneumonia, probably a later manifestation and what first brought our patient to seek medical aid, was not common in the reviewed cases.

Bleeding, either hematemesis or melena, is not usual in uncomplicated achalasia. Though associated with other complications, its appearance with achalasia demands a careful search for carcinoma. The presence of secondary anemia requires the same attention.

Weight loss has been consistent in all cases and must not be explained carelessly as being due to the existing disease.

The major pitfall in the diagnosis is dysphagia, this sign being cardinal in both carcinoma and achalasia. In the former, we have believed it to be due to the mechanical influence of the progressive narrowing of the lumen of the esophagus. We were surprised to find that annular carcinomatous involvement in achalasia was rare, recognizing that the distance "traveled"

VIDENCE CINOMA	ESOPHAGOSCOPY	LOCATION OF TUMOR	SURGERY	SURVIVAL	COMMENT
tion, e third	Ulcerating growth; not annular	Junction middle and distal thirds	Esophagogastrectomy	2 days	Epidermoid tumor
	Fungating mass; not annular	Proximal part of distal third	Not stated	Not stated	Squamous-cell, grade I
	Fungating mass; not annular	Upper middle third	Not stated	Not stated	Squamous-cell
	"Large ulcerating lesion"; not annular	Distal third	None; irradiation	9 plus months	Squamous-cell, grade I
	"Ulcerating and bleeding mass"; ? annular	Middle third	None; irradiation	10 months	Squamous-cell, grade I
	Ulcerating growth; not annular	Middle third	Gastrostomy	15 days	Associated carcinoma cardia; squamous-cell in type as was esophageal lesion
	Not done	Middle third	Heller—cardiomyotomy	Less than 30 days	Squamous-cell carcinom
ne	Not done; tumor ⅞ths of circumference on post mortem	Distal third	Jejunostomy	7 days	Low-grade squamous-ce carcinoma
	Ulcerating lesion	Middle third	Resection with esophagogastrostomy	Died of recurrence at 5 months	Epidermoid carcinoma, grade III; *with meta tases to upper gastr nodes*

by the tumor to accomplish complete encirclement would be many times greater. Still, with complete encirclement, many patients exhibited an unrelenting dysphagia with loss of a previously intermittent character. This, or an advancing dysphagia after certain stability in its severity, we believe to be of diagnostic significance.

If food stasis or peptic regurgitation was a constant factor in mucosal neoplasia of the esophagus, one would expect to find carcinoma in the distal third of the esophagus. Actually, most cases showed malignant changes in the middle third of the esophagus or at the extremities of this segment.

One must expect that, unless an early diagnosis can be made, involvement by contiguous growth will occur before lateral submucosal growth will augment dysphagia or before intramural growth will manifest bleeding. The surgical cure of primary carcinoma of the esophagus is poor because of early invasion of adjacent vital structures; the cure rate of carcinoma in achalasia can be expected to be poorer.

Except in one instance, no direct surgical attack has been made in the cases reviewed. Sicher's[9] patient, who had one of the few lesions of the distal esophagus, was subjected to resection and esophagogastrostomy but survived only two days.

We subscribe to resectional surgery in these cases, if possible, on the same grounds that we subscribe to resectional surgery in "primary" esophageal carcinoma, particularly since irradiation offers nothing for the palliation of achalasia. The emotional reward from a patient who can swallow again buffers the moral uncertainty plaguing the surgeon who must do major palliative surgery.

We agree with Gore and Lam[2] that a right thoracotomy is desirable and utilized a technique similar to that described by DeBakey and Cooley.[11]

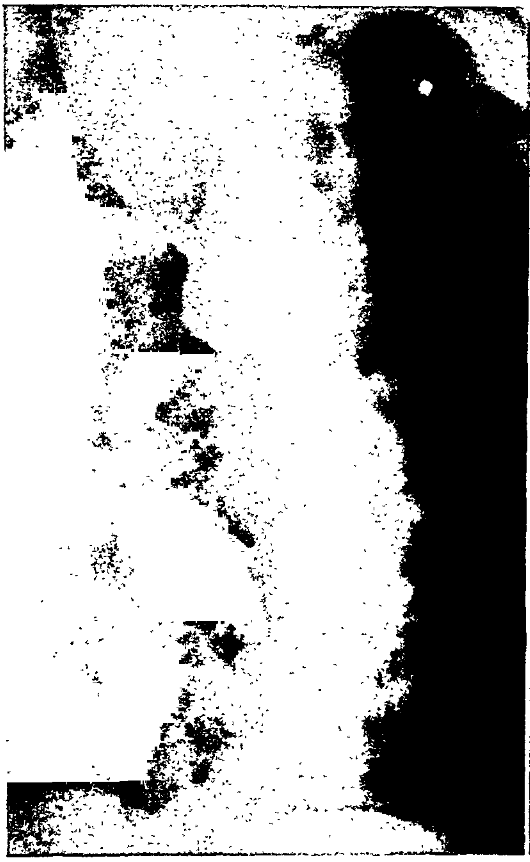

Fig. 1.

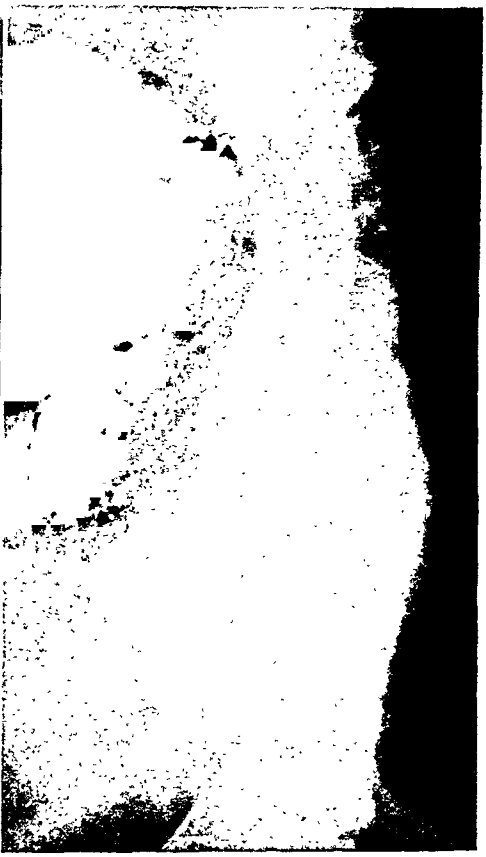

Fig. 2.

Fig. 1.—Achalasia of five years' duration with recent unrelenting dysphagia. Note annular constriction with fluid level distally.

Fig. 2.—Postoperative barium study. Stomach in right hemithorax, less dilatation of proximal esophagus.

SUMMARY

1. Detailed published cases of carcinoma of the esophagus complicating achalasia have been reviewed.

2. Bleeding or progressive unrelenting dysphagia in achalasia warrants a careful search for carcinoma.

3. The need for periodic esophagoscopy and esophagograms is again stressed.

Addendum: Since this paper was submitted for publication, we have noted an article by Klayman (M. I. Klayman, Ann. Int. Med., 43: 33-44, 1955) describing two patients with long-standing cardiospasm whose subsequent carcinoma was proved by exfoliative cytology, the possibility being suspect in only one of these.

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A TECHNICAL AID IN DILATATION OF ESOPHAGEAL STRICTURE

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DILATATION of a benign stricture of the esophagus is a simple procedure in most instances and is deserving of little comment. There is little disagreement as to the safest methods to be employed. Blind methods are unsafe and are not to be recommended. Dilatation under direct vision or over a taut-string guide will prevent the creation of a false passage and all its sequelae. I, personally, prefer the latter since the method is simple and dilatation can be extended to the full normal diameter as indicated.

The passage of a string with a small lead shot attached would, indeed, appear to be a simple solution to the problem. There are instances, however, where the stricture is extremely narrow and there is a definite shelf or ledge just above the opening where the lead shot may "hang up" and pass with extreme difficulty. It occurred to me that a segment of fine watch chain or charm bracelet chain might be substituted for the lead shot. This has proved to be a most effective means of passing the guide string when a lead shot fails to pass. Fig. 1 shows the watch chain and a length of string. Fig. 2 shows the chain at proper level for adequate anchorage of the guide string.

A survey of the available literature did not disclose a report of a similar method. The simple technical aid described here has been most useful in a number of instances and is recommended to facilitate the passage of a guide string in a difficult case.

From the Veterans Administration Hospital, Lincoln, Neb.
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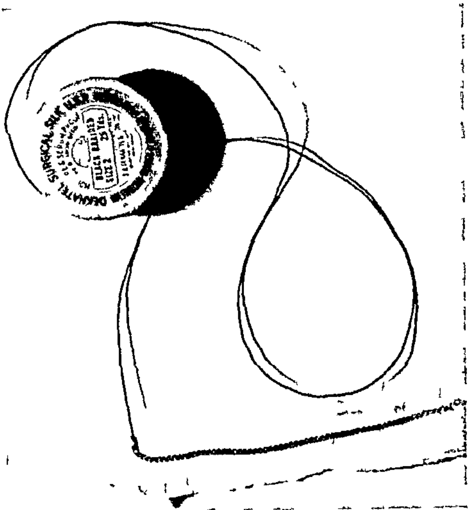
*Chief, Surgical Services and Thoracic Surgical Section, VA Hospital, Lincoln, Neb.

Fig 1



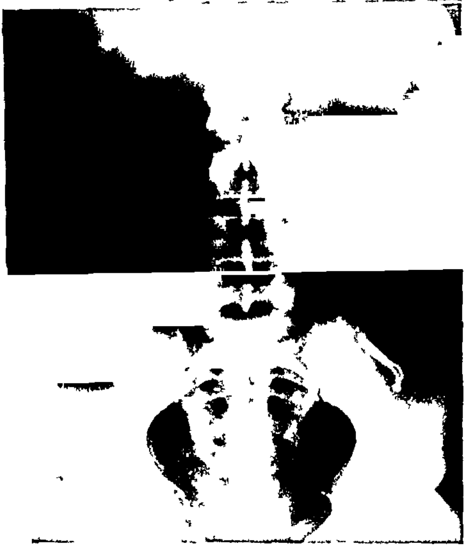


Fig 2

Fig 1 —Heavy silk string attached to fine chain
Fig 2 —String and chain in proper position

SURGICAL MANAGEMENT OF CONGENITAL TRACHEO-ESOPHAGEAL FISTULA AND ESOPHAGEAL ATRESIA

First Report of Patient Surviving Primary Operative Correction of Near Total Thoracic Esophageal Atresia

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PRIOR to 1939, all forms of therapy for tracheo-esophageal fistula and esophageal atresia failed. No infant with esophageal atresia survived. Ladd[1] of Boston and Leven[2] of St. Paul, in 1939, independently, obtained survival in infants with esophageal atresia and distal fistula by closing the fistula and performing cervical esophagostomy and gastrostomy. In an infant treated by this method, the end result was compatible with life. However, the patient had a salivary fistula, had to be gastrostomy fed and the future held multiple-staged, often unsuccessful, operative procedures designed to establish some form of esophageal continuity. The expense was astronomical. Even in the few successful cases, the result was usually a malnourished child, unsightly in appearance.

Cameron Haight[3] of Ann Arbor, Michigan, in 1941, first established esophageal continuity successfully in a one-stage operative procedure by primary anastomosis of the proximal and distal segments. This can almost always be accomplished even in patients with nearly total thoracic esophageal atresias.

There are many combinations of atresia with and without fistula, but about 90 per cent are of the type of proximal atresia with distal tracheo-esophageal fistula. Early diagnosis of tracheo-esophageal fistula and esophageal atresia is essential to successful surgical therapy. One can hardly expect a reasonable surgical mortality if he is operating upon an infant five or six days old who is dehydrated, starved, and suffering from extensive chemical and bacterial pneumonia.

The cardinal symptoms are the baby's apparent excessive salivation and uniform respiratory distress when any attempt at feeding is made. Obviously, the blind proximal pouch results in saliva, water, or formula being aspirated or drooled from the mouth. The diagnosis is confirmed, when suspected, by passage of a soft rubber No. 10 or 12 F. catheter through the mouth into the pharynx and proximal esophagus. Of course, the catheter meets an obstruction in the esophagus. Contrast radiographic studies should be made for further confirmation and to exhibit the rare proximal segment fistulas. The presence or absence of gas shadows in the stomach and small bowel is observed. The degree of pneumonitis, as evidenced by pulmonary infiltration, is also estimated.

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About 25 per cent of these infants are less than 5 pounds in weight.[4] Those untreated die in approximately seven to fourteen days of dehydration, starvation, and pneumonia. The gastric secretion in the first few hours of life is highly acid, and the chemical pneumonia it produces on gaining entrance to the tracheo-bronchial tree through the distal fistula is added to that pneumonia already present from aspirated saliva and other liquids. Most of these babies have no other important associated anomaly. Those that do, have congenital heart disease, imperforate anus, and intestinal atresias and stenoses occurring in that order of frequency.[4]

Immediate operation should be performed except in those patients in whom a delay in diagnosis occurred. Appropriate time should be expended to correct ketosis, dehydration, and pulmonary infection by parental administration of fluid, glucose, vitamins, and antibiotic.

The babies are placed in Fowler's position to minimize regurgitation of acid gastric juice into the trachea. Periodic suctioning from the oropharynx prevents aspiration of mucus. Penicillin and streptomycin are the antibiotics we use by choice. Fluids and blood are administered via a polyethylene tube inserted into the left anterior malleolar vein. In my opinion, the anesthesia of choice for these infants is Pentothal sodium, occasionally supplemented with curare administered through the polyethylene tube inserted into the left anterior malleolar vein, with ventilation maintained by an endotracheal tube. Such controlled respiration makes possible a rapid, precise transpleural operation which may be completed in as little as one to one and one half hours. To date we have not had reason to regret the use of the endotracheal tube.

The retropleural operative approach has long been abandoned by the author since it affords poor exposure and its deficiencies outweigh the advantages. The child is placed in the right chest position. The thorax is entered through the bed of the resected fourth rib (Fig. 1). The mediastinal pleura is entered by dividing the azygos vein. Pleural flaps are carefully preserved for later closure. Just inferior to the azygos vein, the bifurcation of the trachea is easily identified, and the fistulous communication with the distal esophagus dissected free. Care should be taken to avoid injury to the right vagus nerve* which is organized into one or two large trunks and courses obliquely on the lateral aspect of the esophagus and bears little resemblance to the nerve as seen in older subjects. This distal esophagus should be mobilized to the hiatus of the diaphragm, the blood supply enters the esophagus from the medial aspect and is easily preserved. Then the fistulous connection with the trachea is divided near the membranous portion and closed very adequately with a single suture ligature (Fig. 2). By dissecting the membranous portion of the trachea cephalad, the proximal pouch is usually very easily identified as a bulge in the mediastinum. Tenting this structure by a transoral catheter to aid identification is not necessary. The proximal pouch is freed with great care from the posterior membranous portion of the trachea

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*In my experience postoperative apparent pyloric or duodenal obstruction should be considered due to another lesion such as diaphragm atresia or pyloric sphincter hypertrophy rather than loss of gastric tone and pylorospasm due to operative vagal injury. Valuable time may be lost in this erroneous concept (see Case 2).

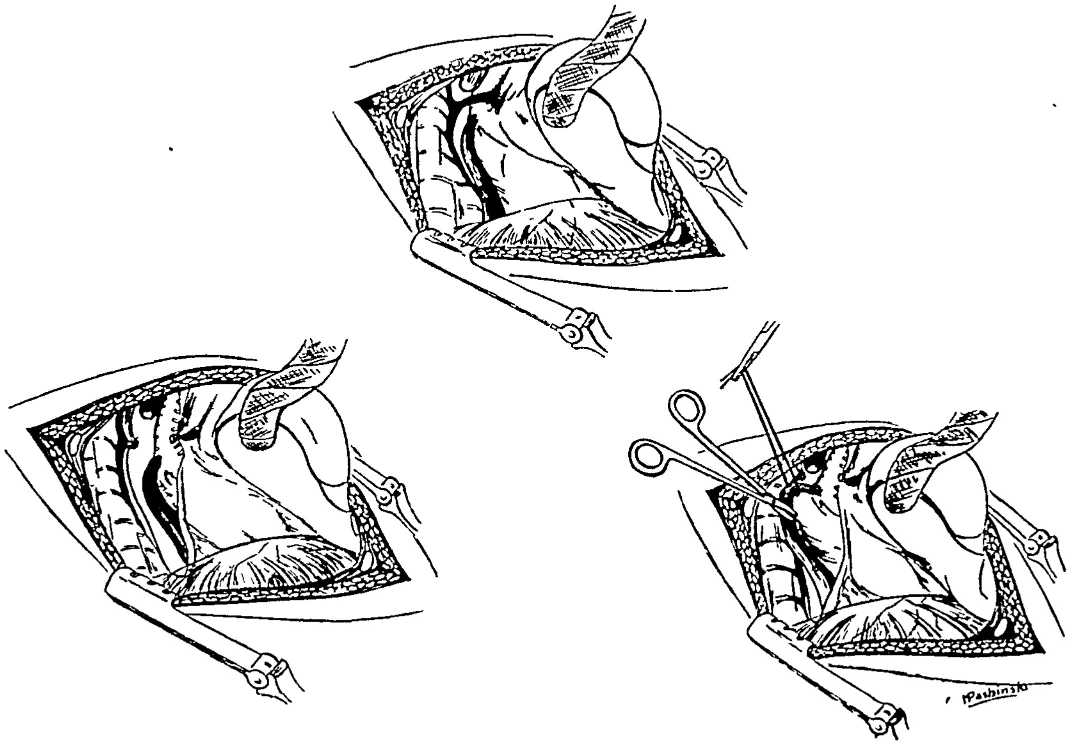

Fig. 1.

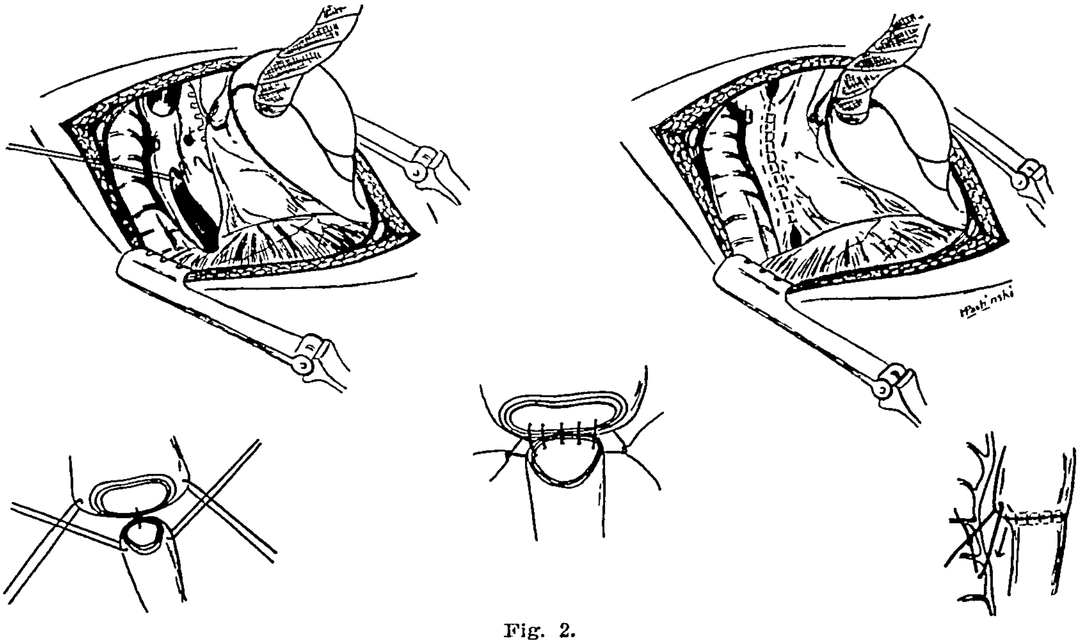

Fig. 2.

since an occasional second fistula may occur at that level. The proximal pouch should be then mobilized far into the neck by blunt and sharp dissection. Significant bleeding is seldom encountered. Devascularization of this pouch has not been a problem.

This will give satisfactory length for a primary anastomosis, which is made in a single layer with interrupted No. 5-0 silk sutures (Fig. 2). The anastomosis should be most meticulously fashioned as this is a critical step in the whole procedure. The proximal and distal ends should be handled only by means of stay sutures and not by tissue forceps. In my opinion, time is wasted on an inferior result when one performs a two-layer anastomosis. Furthermore, the excision of a button of tissue from the proximal segment seems to serve no useful purpose. This often inadvertently affords too large a proximal lumen and may sacrifice much needed length. It fosters mucosa muscularis separation which makes a precise one-layer anastomosis difficult. Suture over an indwelling tube has not been found necessary and results in lost time and increased contamination. After completion of the anastomosis, tension may be considerably relieved by suturing the proximal pouch to the anterior vertebral fascia (Fig. 2). The mediastinal pleural flaps are closed over the anastomosis with a running No. 4-0 plain catgut suture.

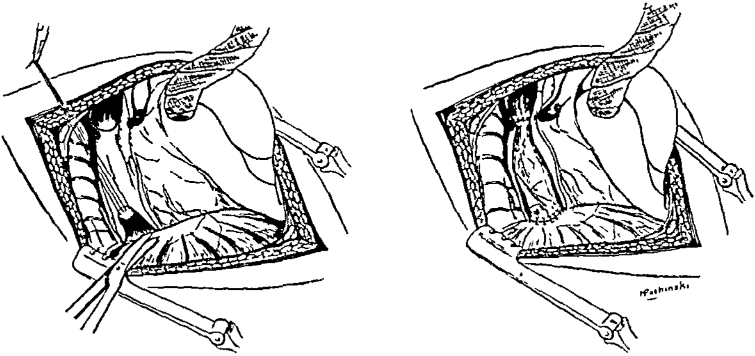

Fig. 3.

The presence of a distal fistula usually ensures a sufficient distal esophageal length for successful primary anastomosis. Large lengths of atresia without fistula may necessitate partial or nearly complete intrathoracic placement of the stomach to accomplish union (Fig. 3).

In my experience, with 1 case of nearly total thoracic esophageal atresia, the following observations were made:

1. A second incision in the rib cage through the bed of the eighth rib was necessary for exposure.

2. After incision of the pleura around the esophageal hiatus, it was easily enlarged to receive the stomach by separating the crural limbs of the opening.

3. Two factors interfered with mobilization; (a) the left gastric vessels which were divided, and (b) the attachments to the spleen on the left side.

Sufficient length was obtained for anastomosis as previously described with gratifying results (Patient C. G.). The wound was closed with interrupted fine catgut sutures, except the skin, and the pleural space drained by a catheter attached to a water seal. Following the surgery and blood replacement, the intravenous polyethylene cannula should be removed by the operator before the patient leaves the operating room. More of these infants have died in the past from overhydration in the postoperative period than from any other single postoperative factor. Postoperative pulmonary edema is usually the result of overhydration rather than any pulmonary injury incident to the anesthesia. These children require very little fluid in the immediate postoperative period. They should be in the oxygen-enriched humidified atmosphere of an incubator; a Stamm type gastrostomy is established in 24 to 48 hours following the thoracotomy. The alternate methods of early feeding without gastrostomy by use of an indwelling esophageal polyethylene tube have resulted in higher morbidity and mortality than gastrostomy. All postoperative fluid needs are well met by subcutaneous fluid until the gastrostomy is established. Penicillin, streptomycin, and vitamin K are routinely given throughout the postoperative period. The infant should be constantly attended with frequent aspirations of the pharyngeal secretions to prevent respiratory complications. If the esophageal anastomosis is under some tension, placing pillows and pads to produce anteflexion of the neck and trunk will relieve tension as flexing a bow loosens its string.[7] Oral feedings are begun on the sixth to eighth day, after satisfactory conditions are demonstrated by a Lipiodol swallow. The gastrostomy tube should be removed only when oral feedings have been well taken for at least one week and radiographs demonstrate an adequate esophageal lumen with no threat of stricture.

Complications which occur in the postoperative period are mediastinitis, pneumonitis, and suture-line leak. The later development of stricture is often due to prior leakage and mediastinitis. Most strictures are well treated by one dilatation. A few may require multiple instrumentations.

Dilatation of postoperative strictures should be done by the operating surgeon. Hubbard and Leven's[6] method of utilizing gum webbing type dilators passed after an attached filiform seems most satisfactory. The willowy filiform may be considerably stiffened by freezing with solid carbon dioxide just prior to use. The operator need allow only a few seconds to elapse after the filiform passes the stricture until it bcomes warmed and sufficiently pliable to allow safe dilatation by the dilator which follows. Dilatation progressively to a No. 16 F. catheter or larger is done depending on the "feel." Stopping short of extensive mucosal tear, as indicated by bleeding, should lessen the tendency to restricture.

CASE REPORTS

CASE 1.—J. C. R. was a 7 pound, 7 ounce, male infant born on Jan. 29, 1953. Eighteen hours after birth, a diagnosis of tracheo esophageal fistula was made by Dr. T. H. Shepherd. Radiographs demonstrated that the orally inserted catheter was arrested at the level of the second thoracic vertebra. There were right upper lobe pneumonitis and atelectasis with mediastinal shift to the right. A normal amount of gas was demonstrated in the stomach, small bowel, and large bowel in the pelvis. Laboratory data were a count of 5.7 million red cells with a hemoglobin of 17 Gm., a count of 12,600 white cells with a left shift. Albuminuria to a degree of 3 plus was found.

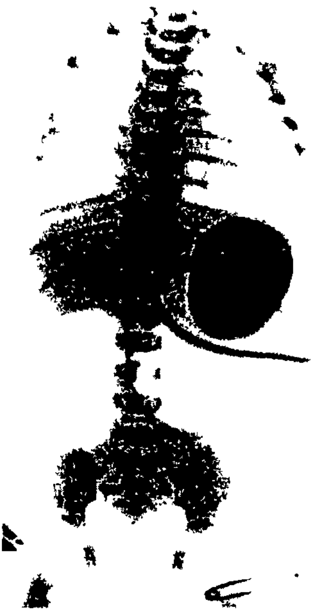
Fig 4.

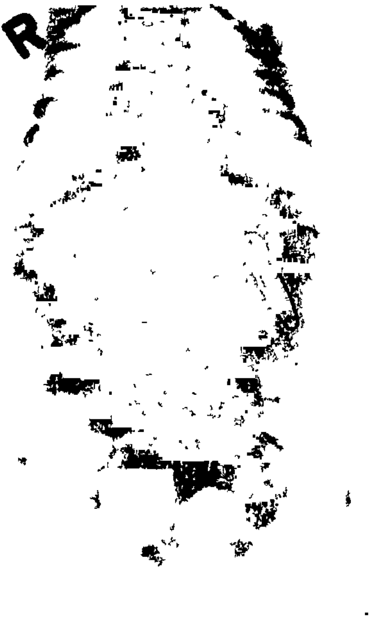

Fig. 5.

Fig. 4—Case 1. (J.C.R.) Lipiodol has been instilled into the gastrostomy tube. The stomach, first, second, and third portion of the duodenum are greatly distended. There is no gas in the lower intestine. The major portion of the right fourth rib has been excised The right lung is well expanded. The previously incomplete duodenal obstruction is now complete

Fig. 5—Case 1. Radiograph made March 16, 1953, the day before the child was discharged from the hospital. Excellent postoperative conditions exist in the thorax and the gastrointestinal gas shadows are within normal limits.

Twenty-three hours after birth, under intravenous Pentothal sodium and curare anesthesia and endotracheal control of respiration administered by Dr. S. T. Hubbard, the chest was entered through a right transpleural approach. A surgical correction of a tracheo-esophageal fistula with division and ligation of the tracheal portion of the fistula and anastomosis of the proximal and distal segments was performed. The pleural space was drained by a No. 10 F. catheter attached to a water seal. The operation was well tolerated. A few days later, a Stamm type gastrostomy was done.

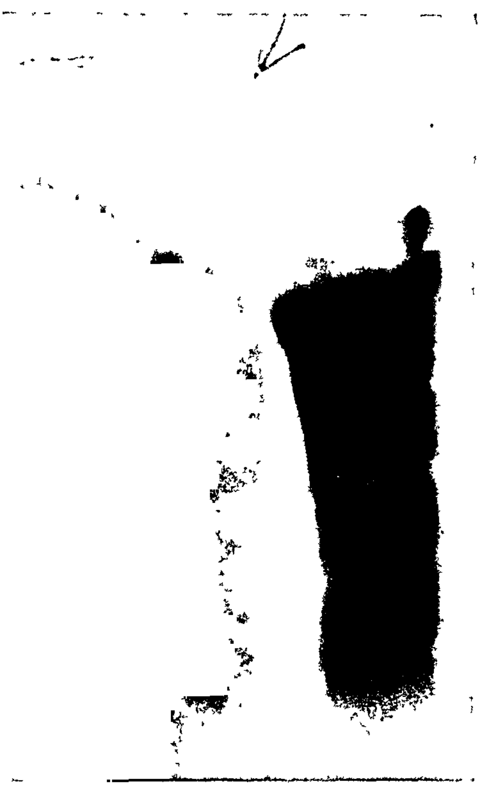

Fig. 6.—Case 1. The Lipiodol esophagram made the day of discharge demonstrates an adequate esophageal lumen with narrowing at the site of anastomosis. There has been no dysphagia since operative correction of the lesion.

Fig. 7.—Case 1. (J.C.R.) The appearance of the child at 17 months of age.

Fig 8

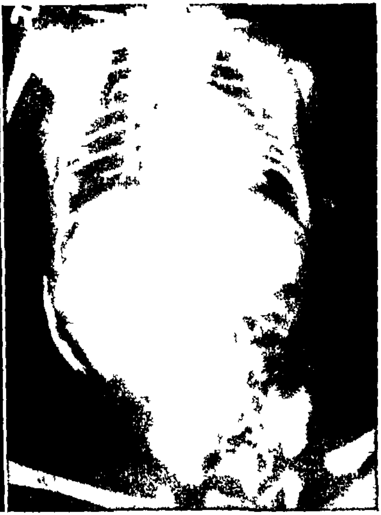

Fig 9

Fig 8—Case 2 (C G) This radiograph taken at the age of 36 hours demonstrates infiltration in the right upper lobe of the lung and absence of any intestinal gas shadows

Fig 9—Case 2 (C G) A radiograph of a Lipiodol swallow demonstrates the esophago-gastric anastomosis and intrathoracic position of the stomach Since only a length of stomach necessary to accomplish anastomosis was placed in the chest encroachment upon the lung with reduction of vital capacity is not a problem

Fig 10—Case 2 (C G) This infant weighed 15 pounds at the age of 11 months and is now well at the age of 20 months Three esophagoscopic examinations with removal of foreign bodies and dilation of an esophagogastric stricture have been necessary Her nutrition is excellent since she eats a normal diet To my knowledge this is the first reported [illegible] of the survival of a patient born with a [illegible] total thoracic esophageal atresia treated by primary esophag[illegible]

Although the child passed stools containing formula, it became apparent that a partial duodenal obstruction had become complete (Fig. 4). On Feb. 10, 1953, a duodenal diaphragm (with an eccentrically placed lumen, 3 mm. in diameter) was excised from the fourth portion of the duodenum. The child recovered and was discharged on March 17, 1953, in excellent condition (Figs. 5 and 6).

He is now 2 years and 5 months old and is, apparently, normal in every respect. There are no esophageal symptoms (Fig. 7).

CASE 2.—C. G. was a 6 pound, 12½ ounce, female infant, 19½ inches long when born on Sept. 30, 1953. A diagnosis of esophageal atresia was made by Dr. W. Lockwood. The child was 36 hours old at the time of diagnosis. Operation was performed at the age of about 40 hours. A right transpleural approach was utilized; the procedure being essentially that illustrated in Fig. 3. Anesthesia was administered by Dr. S. T. Hubbard and consisted of Pentothal sodium and curare with ventilation maintained by endotracheal tube. Anastomosis was fashioned over an indwelling polyethylene tube since the stomach had to be mobilized into the chest to provide gastroesophageal continuity, thus making gastrostomy undesirable. The patient's postoperative course was surprisingly uneventful. The Levin tube was removed on the fourteenth postoperative day and oral feedings were started on the same day. She was discharged on the twenty-eighth postoperative day.

COMMENT

It would seem logical to assume that almost all esophageal atresias could be corrected by a one-stage surgical procedure.

SUMMARY

1. In the past decade and one-half, tracheo-esophageal fistula and esophageal atresia has passed from a uniformly fatal lesion to one successfully treated surgically in a high percentage of the patients. A recovery rate of about 85 per cent is expected in those infants in whom prematurity is not a factor.

2. The report of a patient having survived surgical correction of a nearly total thoracic esophageal atresia has been presented, as well as one of a patient who survived operative correction of both tracheo-esophageal fistula and atresia and excision of a duodenal diaphragm which obstructed the fourth portion of the duodenum.

Excellent assistance in postoperative care of these patients by Drs. W. Lockwood, R. Heskett, and W. Davis is gratefully acknowledged.

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ARTERIALIZATION OF THE CORONARY SINUS IN OCCLUSIVE CORONARY ARTERY DISEASE

IV. CORONARY FLOW IN DOGS WITH AORTICOCORONARY SINUS ANASTOMOSIS OF TWELVE MONTHS' DURATION

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THIS is a continuation of previous studies[1-3] undertaken to investigate the physiologic changes which occur in the coronary circulation following chronic arterialization of the coronary sinus for a period of twelve months.

During the course of our original experiments on the physiology of the coronary circulation in the sinus arterialized hearts of four to eight weeks' duration, we noted a definite protection afforded to the heart against ventricular fibrillation following the acute occlusion of the circumflex branch of the left coronary artery. However, in our subsequent experiments on hearts in which the coronary sinus had been arterialized for six months, we noted a significant loss of this protection.

It was obviously essential to study a series of animals in which the coronary sinus had been arterialized for a prolonged period of time, to determine whether there is a prolonged protection to the heart against the occlusion of a major coronary artery.

Pratt,[4] and Roberts and co-workers[5] originally described the concept of revascularizing the heart in occlusive coronary artery disease by the retrograde perfusion of the myocardium through the coronary sinus. Beck and his associates[6] have developed this procedure to its present two stage technique. The procedure utilizes the placement of a free vein graft between the aorta and the coronary sinus at a first stage, followed by the subsequent partial ligation of the coronary sinus to 2 to 3 mm. at a second stage one month later.

Previously, Hahn and Beck[7] have stated that arterialization of the coronary sinus protected the heart against ventricular fibrillation following the ligation of a major coronary artery. This protection consisted of a reduction in both the mortality rate and the size of the infarct. Eckstein and co-workers[9,10] have also described a protection afforded to the acutely arterialized heart following circumflex coronary artery ligation.

We, too, noted this protection to the heart in our original experiments on dogs in which the coronary sinus had been arterialized for four to eight

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weeks. However, because we could not substantiate this protection in animals in which the coronary sinus had been arterialized for six months, it became necessary to study a series of animals in which the coronary sinus had been arterialized for a truly prolonged period. This study, therefore, comprises a series of animals in which arterialization of the coronary sinus had been performed, and in which the animals were permitted to live a normal kennel existence for a period of one year prior to the institution of the physiologic experiments.

METHOD

Mongrel dogs, whose weight varied between 15 and 18 kilograms, were first prepared using the two-stage operation developed by Beck and his associates. At a first stage, through a left-sided approach, an external jugular vein graft was placed between the aorta and the coronary sinus. Approximately one month later, using a right-sided approach, the coronary sinus was partially ligated over a probe to 2 to 3 mm. Twelve months following the second stage, the physiologic data were obtained. At this time the animals were anesthetized with intravenous pentobarbital sodium, after which artificial respiration was maintained using a pneophore respirator attached to an endotracheal tube with inflated rubber cuff, to which intermittent positive pressure was supplied. The left chest was entered through the fourth intercostal space, and all pleural and pericardial adhesions were freed. The vein graft was inspected and only those animals in which it was patent were selected. An umbilical tape was placed around the graft to facilitate its occlusion at the appropriate moment during the experiment. A needle-tipped polythene catheter attached to a water manometer was inserted into the coronary sinus, through which the coronary sinus pressure and a blood sample for the determination of oxygen content were obtained. The graft was occluded for a period of two minutes, after which the experiment was repeated. The circumflex branch of the left coronary artery was next dissected to its origin and ligated. An incision was made in the wall of the artery, distal to the ligature, through which it was cannulated. A sample of retrograde circumflex coronary arterial blood was collected, simultaneous samples being obtained from the aorta and the pulmonary artery for the determination of oxygen content and saturation data. The minute volume of retrograde blood flow was recorded. At this point, the vein graft was occluded and the experiment was repeated. The peripheral segment of the circumflex coronary artery was permitted to bleed freely for a period of two minutes during which time the retrograde flow volumes were recorded, after which blood samples were collected for determination of oxygen content. This order for the collection of data was decided upon since, in our previous experiments, we noted that the retrograde blood oxygen content gradually increased over a two-minute period before it stabilized.

RESULTS

A. *Retrograde Blood Flow From the Peripheral Segment of the Ligated and Transected Circumflex Branch of the Left Coronary Artery.*—The results are shown in Table I. With the graft open, retrograde blood flow ranged from 1.0 to 12.0 c.c. per minute, with an average of 6.0 c.c. per minute.

Following the occlusion of the aorticocoronary sinus vein graft, the retrograde circumflex artery blood flow ranged from 1.0 to 12.0 c.c. per minute, with an average of 6.6 c.c. per minute. Essentially, the retrograde blood flows remained unchanged in 4 animals and rose slightly in Dogs 2 and 6.

B. *Oxygen Content of the Retrograde Blood Collected From the Peripheral Segment of the Transected Circumflex Artery.*—The results are shown in Table I. The values for the oxygen content of this retrograde blood ranged from 12.9 to 18.7 volumes per cent with an average of 16.3 volumes per cent. Concomitantly, drawn samples of aortic blood ranged from 16.3 to 21.2 volumes per cent with an average of 19.0 volumes per cent.

On careful analysis of these data, it may be observed that the animals, in reality, divide themselves into two groups. In Dogs 2, 4, 5, and 6, the retrograde circumflex blood oxygen content closely approximates that of aortic blood, and is therefore arterial in nature. In Dogs 1 and 3, the retrograde circumflex blood oxygen contents are lower than those of the aortic blood, but are definitely higher than those of the pulmonary artery. They therefore represent a mixture between arterial and a small amount of venous blood. In none of these animals was the retrograde blood oxygen content within the range of the normal coronary sinus blood, as was originally found in the dogs in which true retroperfusion of the coronary sinus existed after one to two months of arterialization. Subsequent to the occlusion of the vein graft, the retrograde blood oxygen content rose farther toward that of aortic blood. These values ranged from 14.6 to 21.2 volumes per cent, and averaged 18.1 volumes per cent. In all the animals, there was a definite rise in the oxygen content of this back flow blood toward the value of the concomitantly drawn sample of aortic blood.

C. *Peripheral Pressure of the Distal Segment of the Transected Circumflex Coronary Artery.*—The results are shown in Table I. Peripheral coronary arterial pressures ranged from 16 cm. of water to 35 cm. of water and averaged 27.5 cm. of water.

Following the occlusion of the aorticocoronary sinus vein graft, the pressure within the peripheral segment of the circumflex coronary artery ranged from 16 to 55 cm. of water, and averaged 34.5 cm. of water.

D. *Mean Coronary Sinus Pressure and Blood Oxygen Content.*—The results are shown in Table I. With the graft open, the mean coronary sinus pressure ranged from 90 to 120 cm. of water and averaged 104 cm. of water. The oxygen content of the arterialized coronary sinus blood ranged from 15.7 to 19.2 volumes per cent with an average of 17.8 volumes per cent. Following the occlusion of the aorticocoronary sinus vein graft, the pressure within the coronary sinus rapidly fell to normal. The pressures now ranged from 4.0 to 6.0 cm. of water and averaged 4.8 cm. of water. The coronary sinus blood oxygen content now ranged from 1.0 to 10.3 volumes per cent with an average of 7.1 volumes per cent.

TABLE I. ARTERIALIZATION OF THE CORONARY SINUS—CORONARY FLOW, AND OXYGEN CONTENT OF BLOOD OBTAINED FROM THE PERIPHERAL SEGMENT OF THE CIRCUMFLEX CORONARY ARTERY—DOGS OF TWELVE MONTHS' DURATION

DOG NUMBER	CIRCUMFLEX CORONARY ARTERY						AORTIC BLOOD				CORONARY SINUS				PULMONARY ARTERY
	GRAFT OPEN			GRAFT CLOSED							GRAFT OPEN		GRAFT CLOSED		
	FLOW (C.C./MIN.)	O_2 CONTENT (VOL. %)	PRESSURE (CM. H_2O)	FLOW (C.C./MIN.)	O_2 CONTENT (VOL. %)	PRESSURE (CM. H_2O)	CONTENT	CAPACITY	SATURATION	PRESSURE	PRESSURE (CM. H_2O)	O_2 CONTENT (VOL. %)	PRESSURE (CM. H_2O)	O_2 CONTENT (VOL. %)	O_2 CONTENT (VOL. %)
1	1.2	12.9	26	1.2	16.8	55	16.3	16.5	98.8	95	90	17.7	5.6	10.1	10.3
2	9.0	18.7	27	11.0	21.2	39	21.2	19.7	107.1	100	100	19.2	4.0	9.9	13.3
3	1.0	13.3	16	1.0	14.6	16	19.8	18.5	107.0	108	106	17.6	4.4	1.0	9.0
4	7.8	18.7	40	7.5	18.2	37	18.7	19.2	97.3	110	110	15.7	6.0		11.0
5	12.0	17.8	35	12.0	20.6	33	20.6	20.4	100.0	100	100	18.9	5.0	11.6	13.7
6	5.0	16.4	21	7.0	17.5	27	17.1	18.1	94.5	120	120	17.7	4.0	10.3	12.3
Average	6.0	16.3	27.5	6.6	18.1	34.5	19.0	18.7	100.8	105	104	17.8	4.8	7.1	11.6

E *Mortality Rate Following Circumflex Coronary Artery Ligation in Animals With Arterialization of the Coronary Sinus of Twelve Months' Duration* — Since we desired to study all specimens pathologically, we selected an arbitrary survival time of four hours, subsequent to the ligation of the circumflex coronary artery, as the criterion for a comparative mortality report Those animals which survived for four hours, following the ligation of the circumflex coronary artery, were classified as survivals We thus continue to utilize the same criteria for survival as was established in our previous experiments In the present series, 2 dogs survived for the prescribed period of time prior to sacrifice Four dogs died of ventricular fibrillation within one hour of the ligation of the circumflex artery This provides a mortality rate of 66 per cent The records of the survivors (Dogs 2 and 5) may be seen in Table I They demonstrated the highest rates of retrograde circumflex arterial flow These flow rates were unchanged following the occlusion of the graft which probably indicates the existence of a more effective coronary collateral circulation

DISCUSSION

The present series of experiments on animals in which the coronary sinus had been arterialized for a period of twelve months concludes an investigation that we have been conducting for the past several years, of the effects of arterialization of the coronary sinus on the coronary circulation Since we have previously investigated the physiologic effects of arterialization of the coronary sinus on the coronary circulation of animals over shorter periods of time, it would be valuable to review these findings

In a series of normal control animals, we have demonstrated a small quantity of intercoronary collateral flow, measuring approximately 1 7 c c per minute This blood was invariably arterial in nature, which demonstrated its origin to be from the neighboring coronary arteries This backflow therefore represented a measure of normal arterial coronary collateral flow

In a series of animals in which the coronary sinus had been arterialized for four to eight weeks, these findings were significantly changed" With the graft functioning, the retrograde circumflex arterial blood flow averaged 12 2 c c per minute, thus representing a significant increase over that found in the normal animals Moreover, this back blood which arose from the peripheral segment of the circumflex coronary artery differed from that of the normal animal since it was now markedly deoxygenated, the oxygen content being within the range of normal coronary sinus blood The desaturation of this blood clearly demonstrated that there was a retroperfusion of the myocardial capillary bed, with extraction of oxygen by the myocardium When the aorticocoronary sinus vein graft was occluded, the retrograde circumflex coronary arterial blood flow fell to 7 1 c c per minute and the oxygen content returned to normal The arterial nature of this blood again signified that it had arisen from intercoronary anastomoses This retrograde coronary arterial blood flow represented a fourfold increase above that normally expected, and therefore represented a significant increase in the intercoronary arterial anastomotic flow

In the series of animals previously reported upon,[3] in which the coronary sinus had been arterialized for six months, the findings were significantly changed from those in the four- to eight-week group. Here, with the graft functioning, the retrograde coronary arterial blood flow averaged 8.3 c.c. per minute. This blood was highly oxygenated, the oxygen content being within the range of concomitantly drawn samples of aortic blood. The arterial nature of this blood pointed toward its origin from neighboring unligated arteries, thus demonstrating an increase in the intercoronary collateral flow. When the aorticocoronary sinus vein graft was occluded, the retrograde circumflex arterial blood flow again averaged 8.7 c.c. per minute. The oxygen content of the retrograde blood was again unchanged and remained arterial in nature. Strikingly, then, in those animals in which the coronary sinus had been arterialized for a period of six months, the retrograde circumflex coronary arterial blood flow could not be altered by the opening or the closing of the aorticocoronary sinus vein graft. Moreover, the oxygen content of this blood was also unaffected by the presence or absence of a functioning vein graft. In short, the significant finding in the series of animals in which the coronary sinus had been arterialized for a period of six months, was that there was no significant contribution to the retrograde circumflex arterial flow by the arterialized coronary sinus. We were able to demonstrate an increase in the intercoronary arterial anastomotic flow, but were unable to demonstrate any significant retroperfusion of the myocardial capillary bed.

The present investigation of the coronary circulation of animals in which the coronary sinus was arterialized for a prolonged period of one year's duration merely serves to reconfirm the findings that have been described in the six-month group of animals. With the graft open and functioning, the retrograde circumflex coronary arterial blood flow averaged 6 c.c. per minute. This blood was oxygenated and within the range of concomitantly drawn samples of aortic blood. There were a few animals in which the oxygen content of the retrograde blood was definitely lower than that of concomitantly drawn samples of aortic blood, but in all the dogs the oxygen content was significantly higher than that of concomitantly drawn samples of pulmonary arterial blood. The oxygen content of the retrograde coronary arterial blood averaged 16.3 volumes per cent as compared to an average aortic oxygen content of 19.0 volumes per cent, and a pulmonary arterial oxygen content of 11.6 volumes per cent. When the aorticocoronary sinus vein graft was clamped, the backflow from the peripheral segment of the circumflex coronary artery averaged 6.6 c.c. per minute, which is insignificantly changed from that observed with the graft open. The oxygen content of this retrograde blood averaged 18.1 volumes per cent; a 2 volume per cent increase over that found with the graft closed.

Significantly, in the present series of animals in which the coronary sinus was arterialized for a period of one year, the retrograde blood flow from the peripheral segment of a ligated and transected coronary artery, and oxygen content of this blood are completely unaffected by the presence or absence of a functioning aorticocoronary sinus vein graft. The arterialized coronary sinus has apparently completely lost contact with the capillary bed.

It might be well to review the comparative mortality statistics compiled during the course of our series of experiments. Originally, when the circumflex branch of the left coronary artery was ligated in a group of normal animals, the mortality rate was 90 per cent. In a second group of animals in which previous nondescript cardiac procedures had been performed, coronary arterial ligation produced a mortality rate of only 66 per cent. In the series of animals in which the coronary sinus had been arterialized for one to two months, we were very impressed by an apparent protection afforded the heart against the onset of ventricular fibrillation following major coronary artery ligation. Here, the mortality rate fell to only 20 per cent. This impression was quickly dispelled by our subsequent experiments on animals in which the coronary sinus had been arterialized for six months. In this group, we were unable to ascertain any protection afforded to the heart against ventricular fibrillation by the presence of the aorticocoronary sinus vein graft. The mortality rate had risen to 66 per cent which resembled that found in the group in which only nondescript cardiac procedures had been performed. This fact was further substantiated by the results in the present twelve month group of animals. Here, the mortality rate following circumflex coronary arterial ligation was again 66 per cent, which is again identical to that of the six-month group of animals and those having had nothing more than nondescript cardiac procedures. These findings again serve to confirm those recently reported by Eckstein and his associates,[10] as well as our own.[3] These workers noted that the retroperfusion of the myocardium, which existed in dogs in which the coronary sinus had been arterialized for four weeks, could no longer be demonstrated after three months. At the end of this time, they too noted that the retrograde circumflex coronary arterial blood again became arterial in nature, and therefore postulated its origin to be from the interarterial collateral circulation. They were able to demonstrate that blood which flowed in retrograde fashion from the peripheral segment of the ligated circumflex coronary artery, probably originated in the anterior descending coronary artery. They were unable to demonstrate any significant effect, by the arterialized coronary sinus, upon the blood arising from the peripheral segment of the ligated and transected coronary artery.

The retrograde circumflex arterial blood flows, as seen in Dogs 2 and 6, Table I, with the graft open and closed, bear comment. Here, the backflow increased following the occlusion of the aorticocoronary sinus vein graft. We have described this increase in retrograde flow following clamping of the graft in previous experiments on animals in which the coronary sinus had been arterialized for six months. Eckstein and his associates[9, 10] have similarly described this increase in several animals in which the sinus had been arterialized for periods longer than three months. At post mortem examination of these hearts, we noted that the ostium of the coronary sinus had not been reduced to 2 mm. Possibly, the orifice of 4 mm. which existed in these animals permitted an arteriovenous fistula of such magnitude that occlusion of the graft, by obviating the fistula, improved the coronary flow of the unligated anterior descending coronary artery, and thereby increased the interarterial collateral flow.

The long-term series of experiments reveal that the favorable effects produced by arterialization of the coronary sinus apparently regress over a prolonged period of time. We first observed this regression at the end of six months, and have definitely reconfirmed it at the end of a year.

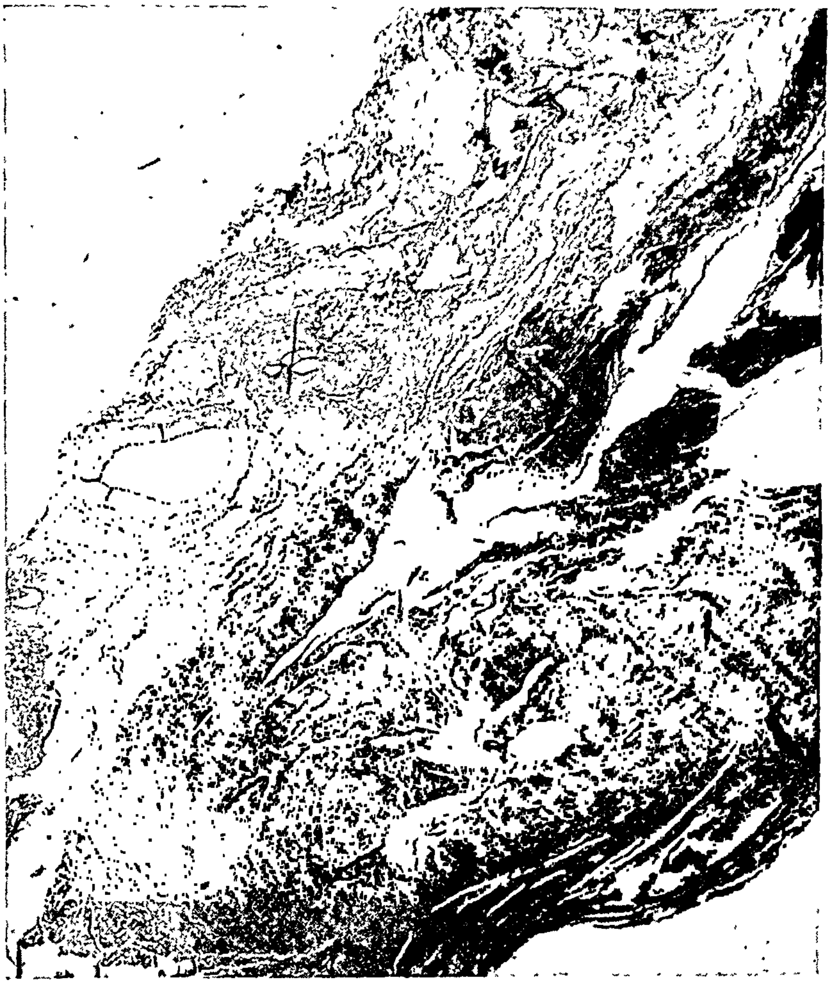

Fig. 1.—Low-power magnification showing a major artery and vein. The artery is normal. The lumen of the vein is obliterated by severe intimal proliferation and thrombosis. Recanalization of the thrombosed lumen of the vein is evident.

The inability to demonstrate any retrograde perfusion of the myocardial capillary bed by physiologic means is further substantiated by the microscopic pathologic findings at the time of the post-mortem examination. There is a marked thickening of the coronary sinus itself due to intimal proliferation. Moreover, all of the major tributaries to the coronary sinus demonstrate a severe degree of intimal proliferation. The lumina of these vessels are so markedly encroached upon, that they may be considered to have been completely occluded by this process (Figs. 1 and 2). Apparently, prolonged periods of coronary sinus hypertension produce a severe degree of intimal proliferation within the small and large veins which empty into it. This intimal proliferation effectively occludes these vessels and subsequently prevents any retroperfusion of the myocardial capillary bed.

Thus, since the original retroperfusion of the myocardial capillary bed is obviated after a period of time because of the high head of pressure within the coronary sinus and its venous tributaries, it is apparent that the end result *of the procedure is to stimulate the development of the intercoronary* collaterals. It strikes us that, since the prolonged effect of arterialization of the coronary sinus is one of increasing the intercoronary arterial anastomotic flow, it may be possible to attain this result by a more simple procedure. We are therefore instituting an evaluation of simpler procedures designed to increase the intra-extracoronary collateral flow.

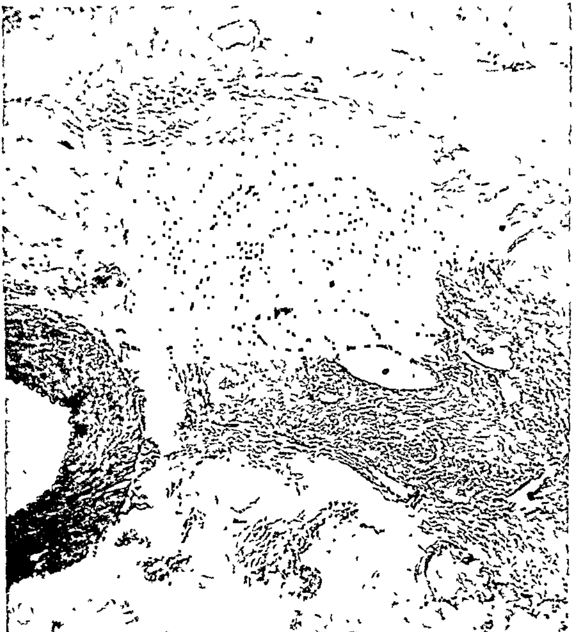

Fig. 2.—High-power magnification of the vein reveals old thrombosis with organization and recanalization of the lumen.

SUMMARY AND CONCLUSIONS

1. The retrograde blood flow and oxygen content of the blood from the peripheral segment of a ligated and transected circumflex artery have been determined in dogs in which the coronary sinus has been arterialized for a period of one year. These determinations have been performed with the graft open and closed.
2. With the graft open, coronary sinus pressure is within the range of that of the systemic circulation and the coronary sinus blood is of high oxygen content.
3. When the graft is occluded, the coronary sinus pressure and blood oxygen content revert to those of normal coronary sinus levels.
4. With the graft open, retrograde circumflex coronary arterial blood flow averages 6 c.c. per minute. This represents a threefold increase over that expected in the control animal, but is less than that previously found in the two- and six-month groups of animals. This blood is arterial in nature, approximating the oxygen content of aortic blood. The retrograde flow is probably due to an increase in the intercoronary arterial collateral circulation.
5. Occlusion of the aorticocoronary sinus vein graft does not significantly alter these findings. This indicates that after one year, the vein graft no longer contributes to the interarterial collateral circulation and is dispensable.
6. The mortality rate following circumflex coronary arterial ligation is 66 per cent in the one-year group of animals, which is unchanged from that previously reported in our six-month group of animals.
7. Retroperfusion of the myocardial capillary bed could not be demonstrated in this group of animals.

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MEASUREMENTS OF THE UNILATERAL RESIDUAL VOLUME*

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INTRODUCTION

IT HAS been among our objectives over the past three years to evaluate the effects of pulmonary resection [1-3] Included in the studies has been the measurements of the total capacity of the lungs Because such data present an *inaccurate description of the effects of procedures affecting the two lungs unequally*, we have sought to measure the total capacity of each lung individually in association with bronchospirometry

References to similar attempts at measuring the unilateral residual volume are few Bjorkman[4] has recorded the complete unilateral and bilateral volumetric data of 2 patients and Gaensler and Cugell[5] have listed such figures for 1 patient Although a considerable experience with this procedure is indicated by the reports of Frenckner,[6] no figures are listed by which the unilateral technique can be compared with an established method for measuring bilateral volumes

The present brief report includes the unilateral and bilateral total lung volumes of 9 patients Six were selected because the pulmonary lesion was thought to affect the distribution or dimensions of the lung volumes minimally, their data are therefore considered to approximate the normal distribution Three patients were chosen in order to illustrate the application of the technique where the conventional bilateral figures are of equivocal value

CLINICAL MATERIAL

The 6 "control" patients (Table I) were men with pulmonary tuberculosis restricted either to the apex of the right or the left upper lobe or to the superior segment of the right lower lobe (Patient 0586) Each was a candidate for segmental resection at the time of study The youngest patient was 18 and the oldest 39 years of age The vital capacity was normal in each instance

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TABLE I. UNILATERAL AND BILATERAL TOTAL LUNG VOLUMES IN SIX MEN WITH NORMAL UNILATERAL AND BILATERAL VITAL CAPACITIES

PATIENT NUMBER		0490	0500	0523	0586	0612	0627
AGE (YEARS)		23	23	39	18	34	21
B.S.A. (SQ. M.)		1.67	2.12	1.70	1.89	1.89	1.96
DIAGNOSIS		Mod. tbc LUL	Min. tbc RUL	Mod. tbc LUL	Min. tbc RLL	Min. tbc RUL	Mod. tbc LUL
VITAL CAPACITY	Rt. c.c.	2462	2774	2416	2850	2699	2662
	Lt. c.c.	1928	2328	2086	2508	2451	2143
	Sum. c.c.	4390	5102	4502	5358	5150	4805
	Bilat. c.c.	4832	5979	4622	5760	5294	5469
	Rt. %	56	54	54	53	52	55
	Lt. %	44	46	46	47	48	45
RESIDUAL VOLUME	Rt. c.c.	1198	1032	732	756	1418	528
	Lt. c.c.	1078	745	634	807	1298	302
	Sum. c.c.	2276	1777	1366	1563	2716	830
	Bilat. c.c.	2127	1660	1572	1490	2935	851
	Rt. %	53	58	54	48	52	64
	Lt. %	47	42	46	52	48	36
TOTAL LUNG CAPACITY	Rt. c.c.	3660	3806	3148	3606	4117	3190
	Lt. c.c.	3006	3073	2720	3315	3749	2445
	Sum. c.c.	6666	6879	5868	6921	7866	5635
	Bilat. c.c.	6959	7639	6194	7250	8229	6320
	Rt. %	55	55	54	52	52	57
	Lt. %	45	45	46	48	48	43
FUNCTIONAL RESIDUAL CAPACITY	Rt. c.c.	2094	1645	1268	1618	2160	1196
	Lt. c.c.	1729	1335	1045	1669	1995	948
	Sum. c.c.	3823	2980	2313	3287	4155	2144
	Bilat. c.c.	3537	3251	2528	3445	4495	2282
	Rt. %	55	55	55	49	52	56
	Lt. %	45	45	45	51	48	44
MIXING INDEX	Rt. % N_2	1.72	2.16	1.72	0.96	4.41	5.96
	Lt. % N_2	8.34	2.08	2.17	1.22	7.51	0.47
	Bilat. % N_2	2.52	2.42	0.52	0.60	2.13	0.72
$\left(\frac{\text{R.V.}}{\text{T.L.C.}}\right) \times 100$	Rt.	33	27	23	21	34	17
	Lt.	36	24	23	24	35	12
	Bilat.	31	22	25	21	36	13
$\left(\frac{\text{F.R.C.}}{\text{T.L.C.}}\right) \times 100$	Rt.	57	43	40	45	52	37
	Lt.	58	43	38	50	53	39
	Bilat.	51	43	41	48	55	36

Two of the other 3 patients (Table II, Figs. 1 and 2) had been treated surgically for pulmonary disease. Patient 0003 had had resections of seven bronchiectatic bronchopulmonary segments: the right middle lobe, the lingula, and the basilar segments of the left lower lobe. A postoperative empyema had resulted in fixation of the left hemidiaphragm (Fig. 1*A*). Patient 0866 had bilateral tuberculosis. A seven-rib thoracoplasty had been performed in an attempt to control cavitary disease on the left side (Fig. 2*A*). The ninth patient, Number 1026 (Table II, Fig. 3), also with bilateral pulmonary tuberculosis, was studied after spontaneously developing a right phrenic nerve paralysis (Fig. 3*A*).

METHODS

The study of each patient included a measurement of the total capacity of both lungs as well as of each lung individually. In obtaining the conven-

TABLE II. UNILATERAL AND BILATERAL TOTAL LUNG VOLUMES IN THREE MEN WITH ABNORMALLY UNEQUAL UNILATERAL VOLUMES

PATIENT NUMBER		0003	0866	1026
AGE (YEARS)		36	39	29
B.S.A. (SQ. M.)		2.21	1.79	1.93
DIAGNOSIS		Bilat. resection bronchiectasis, Postop. empyema, Lt.	Bilat. tbc., Lt. thoracoplasty	Bilat. tbc., Apical disease, Rt. phrenic paralysis
VITAL CAPACITY	Rt. c.c.	2553	1566	654
	Lt. c.c.	1451	358	2187
	Sum c.c.	4004	1924	2841
	Bilat. c.c.	4026	1944	3293
	Rt. %	64	81	23
	Lt. %	36	19	77
RESIDUAL VOLUME	Rt. c.c.	609	609	287
	Lt. c.c.	1484	353	688
	Sum c.c.	2093	962	975
	Bilat. c.c.	2305	890	1005
	Rt. %	29	63	29
	Lt. %	71	37	71
TOTAL LUNG CAPACITY	Rt. c.c.	3162	2173	941
	Lt. c.c.	2935	711	2875
	Sum c.c.	6097	2884	3816
	Bilat. c.c.	6331	2834	4298
	Rt. %	52	75	25
	Lt. %	48	25	75
FUNCTIONAL RESIDUAL CAPACITY	Rt. c.c.	1141	1108	437
	Lt. c.c.	1914	398	1004
	Sum c.c.	3055	1506	1441
	Bilat. c.c.	3070	1458	1564
	Rt. %	37	74	30
	Lt. %	63	26	70
MIXING INDEX	Rt. % N	1.79	0.90	2.40
	Lt. % N_2	1.01	3.88	0.90
	Bilat. % N	2.61	1.88	1.01
$\left(\frac{\text{R.V.}}{\text{T.L.C.}}\right) \times 100$	Rt.	19	28	30
	Lt.	51	50	24
	Bilat.	36	31	23
$\left(\frac{\text{F.R.C.}}{\text{T.L.C.}}\right) \times 100$	Rt.	36	51	46
	Lt.	65	56	35
	Bilat.	48	51	36

tional bilateral figures, the functional residual capacity was measured twice, using a modification of the Darling method. Duplicate analyses agreed within 10 per cent, the mean of these two values appears in the tables. The bilateral index of intrapulmonary mixing listed in the tables is similarly a mean of the nitrogen concentrations in the "alveolar" air samples delivered at the end of each seven minute period of oxygen breathing. The vital capacity and its subdivisions were recorded in triplicate, the best of the three tracings being used in computing the residual volume and the total lung capacity.

Bronchospirometry was performed under 1 per cent Pontocaine local anesthesia after premedication with Nembutal, atropine, and morphine. The catheter (Carlens[8]) was introduced, usually over a flexible sound, into the upper larynx under mirror visualization. It was gently rotated through the

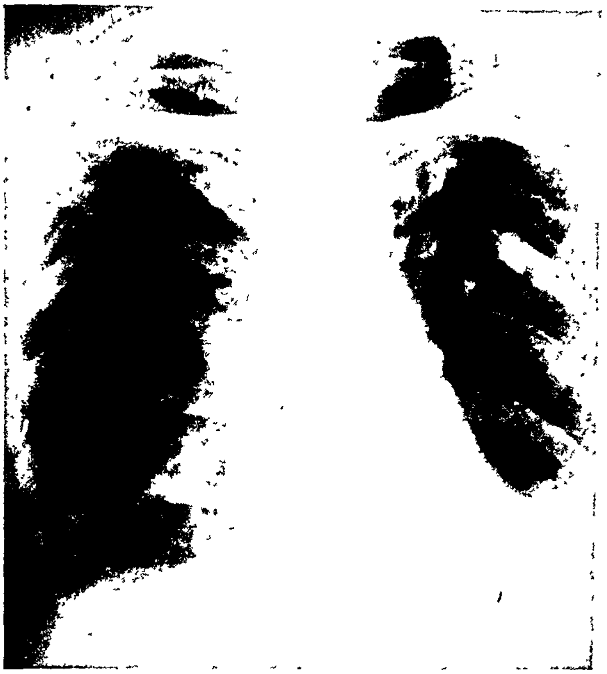

Fig. 1*A*.—Patient 0003. Bilateral resections for bronchiectasis. Postoperative chest roentgenogram. Note evidence of previous empyema on the left with pleural thickening and elevation of diaphragm. At fluoroscopy the left hemidiaphragm was found to move minimally on forced inspiration and expiration, the left rib cage was relatively fixed and the left lung field was more radiolucent than the right.

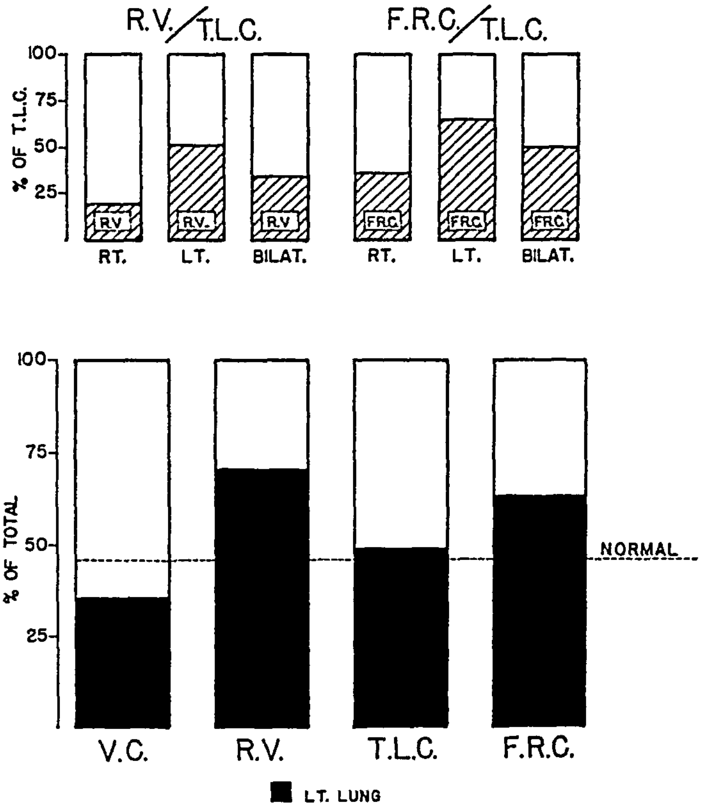

Fig. 1*B*.—Patient 0003. Unilateral and bilateral lung volumes and volumetric ratios. Note: (1) Although the total lung capacity is normally distributed, the other compartments are not: the vital capacity on the left is low but the residual volume on this side is high. (2) The ratio of the bilateral residual volume to the total lung capacity is normal in spite of gross abnormality of this ratio on the left.

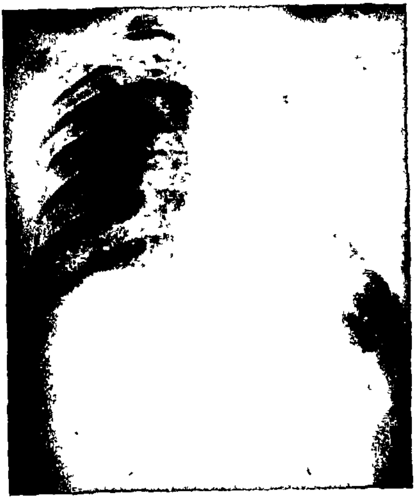

Fig. 2A.—Patient 0866. Bilateral pulmonary tuberculosis, left thoracoplasty.

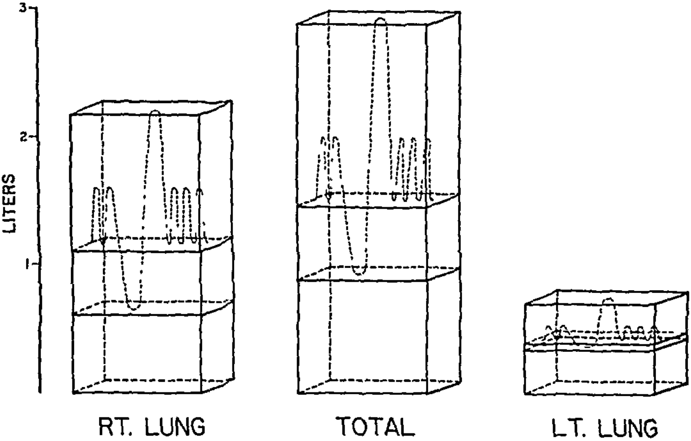

Fig. 2B.—Patient 0866. Unilateral and bilateral lung volumes. Note the magnitude of the differences in the distributions of the various volumetric compartments. These are identified by name in Fig. 3B.

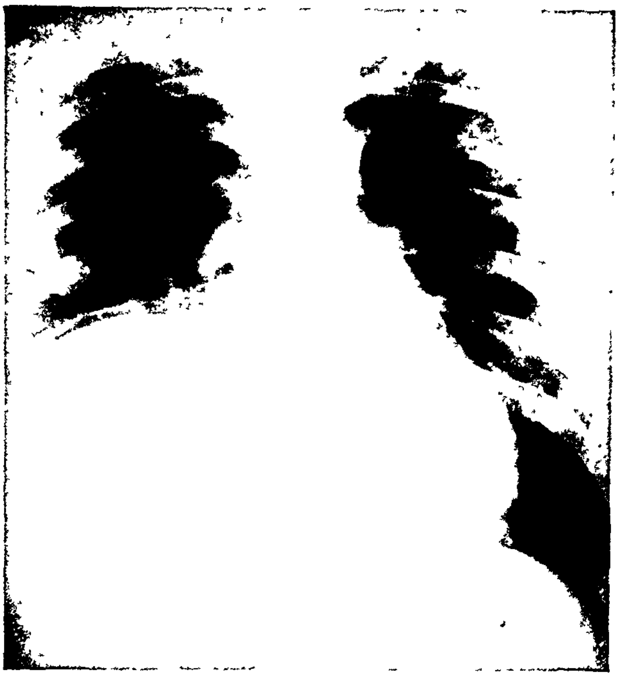

Fig. 3A.—Patient 1026. Bilateral pulmonary tuberculosis, spontaneous right phrenic nerve paralysis.

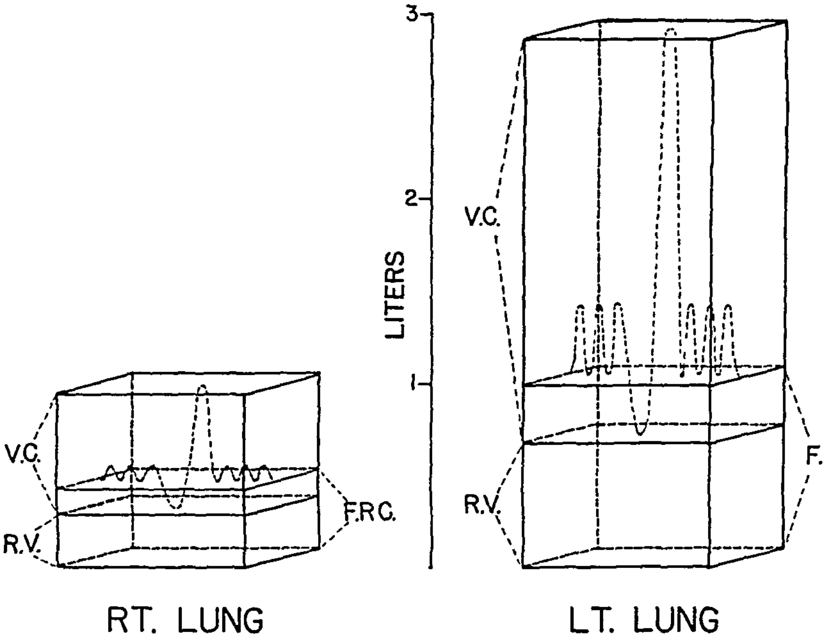

Fig. 3B.—Patient 1026. Unilateral lung volumes. Note the practically symmetrical reduction in the various subdivisions of the total capacity of the right lung. V.C.—vital capacity, R.V.—residual volume, F.R.C.—functional residual capacity.

glottis after removal of mirror and sound and was positioned with the assistance of a fluoroscope. Its spur was not tied down for passage into the trachea.

The two lumina of the catheter were initially coupled to a bronchospirometer, the bells of which were filled with room air. After a satisfactory three minute tracing of oxygen consumption and tidal respiration had been recorded, the patient was instructed to deliver his expiratory reserve and, following restoration of the mid position, his inspiratory capacity. The bronchospirometer was then uncoupled and each side of the catheter was fitted to a mobile unit for measuring the unilateral functional residual capacity. Each unit consisted of a vertically adjustable table on which lay a Douglas bag for collecting expired air. The coupling to the patient led into a valve which could be turned either into a circuit incorporating this bag with one-way valves and a source of oxygen or into an arm communicating optionally with the room or with an underwater trap for collecting the sample of "alveolar" air. A check valve prevented aspiration of the water in the trap. The patient was switched from room air into the oxygen circuit at the end of what was judged to be a normal expiration. The expired air was collected for nine minutes, during which time nitrogen was displaced from the alveolar trap circuit by flushing with oxygen. At the end of the nine minutes, the patient was instructed to inspire normally and hold his breath while the valves were turned to the alveolar traps. He then expired slowly and fully through the trap, the valve being turned to room air just prior to inspiration.

In most instances, it was possible as a final step to obtain a second bronchospirometric tracing as a check on the location of the catheter. When a change in position was indicated, the determination was discarded.

The volume of the catheter lumina, connecting tubes, and valves was 130 c.c. Since the anatomic dead space for which this volume was substituted approximates 150 c.c., no correction was included. The figure for nitrogen excretion incorporated in the equation for calculating the functional residual capacity was divided between the two lungs according to the distribution of the oxygen consumption.

Nitrogen analyses were performed in Van Slyke manometric equipment. All lung volumes were converted to body temperature, ambient pressure, saturated with water vapor (BTPS).

RESULTS (TABLES I AND II)

In these 9 patients, the sum of the *functional residual capacities* of the two lungs differed from the mean of the duplicate bilateral values by less than 10 per cent.* This level of agreement was found for the *residual volume* in all but 1 of the patients (No. 0523, 13.1 per cent), with respect to the *vital capacity* with three exceptions (No. 0500, 14.7 per cent, No. 0627, 12.1 per cent, and No. 1026, 13.7 per cent), and for the *total lung capacity* with two exceptions (No. 0627, 10.8 per cent, and No. 1026, 11.2 per cent). Percentile correspondence of the two techniques with respect to residual volume and func tional residual capacity, therefore, fell well within the range found for vital capacity. Since the former two compartments are considerably smaller than the latter, the actual volumetric differences were considerably less.

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*Calculated by dividing the difference between bilateral and sum of unilateral volumes by the bilateral value.

Mean values for the distribution of the various compartments between the two lungs in the "normal" group (Table I) were nearly identical, being 54 per cent on the right and 46 per cent on the left except for the residual volume (55 per cent right, 45 per cent left.) Variations in the distribution of the residual volume ranged from 48 per cent to 64 per cent for the right lung. Corresponding figures for the functional residual capacity and vital capacity, respectively, were 49 per cent to 56 per cent and 52 per cent to 56 per cent.

In the other 3 patients (Table II) the distribution of the lung volumes differed markedly from the normal. Patient 0003 had 36 per cent of the vital capacity on the left but 71 per cent of the residual volume on this side (Fig. 1*B*). Corresponding figures for Patient 0866 were 19 per cent and 37 per cent, respectively (Fig. 2*B*). In Patient 1026, on the other hand, all compartments were proportionately reduced on the side of phrenic paralysis (Fig. 3*B*).

Among the six "normals," differences in excess of 4 per cent between a unilateral and the bilateral R.V./T.L.C. or F.R.C./T.L.C.* were found for 3 of the 12 lungs (No. 0490—left R.V./T.L.C., left and right F.R.C./T.L.C.; No. 0500—right R.V./T.L.C.). There were differences of this magnitude between the 2 lungs of the same individual in two instances (No. 0627—R.V./T.L.C.; No. 0586—F.R.C./T.L.C.). As would be expected, the variations between the 2 lungs and between 1 lung and the bilateral value were large in the 3 abnormal patients.

The mixing index was grossly different (more than 2 volumes per cent nitrogen) in four of eighteen comparisons between a unilateral and the mean of two bilateral values (Patients 0490, 0612, and 0627). The unilateral concentrations were usually somewhat higher than the bilateral for reasons which will be discussed.

DISCUSSION

Measuring the functional residual capacity of each lung individually by the method employed is time consuming and technically difficult. Eight nitrogen analyses, including unilateral and bilateral determinations, are needed for each complete study. The usual problems of satisfactory bronchospirometry, in particular the presence of secretions, are compounded by the added time required. The impediments to normal breathing created by the presence of the catheter result in a high incidence of poor checks between unilateral and bilateral functional residual capacities. Since a reasonable agreement by the two techniques is essential for evaluating a given unilateral analysis, data must be discarded frequently.

A number of factors are helpful in increasing the yield of acceptable studies. Primary among these are selecting cooperative patients with dry bronchial trees and developing a smooth, rapid routine for the various steps involved.

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*R.V./T.L.C. = ratio of the residual volume to the total lung capacity; F.R.C./T.L.C. = ratio of the functional residual capacity to the total lung capacity.

Difficulties notwithstanding, however, it is evident that knowledge of the unilateral residual volume is occasionally essential to an intelligible analysis of volumetric status. The usual bilateral and unilateral measurements of Patient 0003, for example, fail to suggest the extent of the abnormality resulting from fixation of the left diaphragm in a depressed position. Assessing the effects of phrenic interruption, as in Patient 1026, and of various forms of unilateral collapse or resection therapy requires a knowledge of unilateral residual volume. It should be noted that the elevation of the right diaphragm and consequent low functional residual capacity for the right lung in Patient 1026 (Figs. 3*A* and 3*B*) is rarely seen following therapeutic phrenic nerve interruption, a procedure which in the absence of pneumoperitoneum usually fails to change the unilateral mid position.[6, 7]

The limitations imposed by a knowledge only of the bilateral residual volume and the unilateral vital capacity, apparent in evaluating the effects of resection in Patient 0003, are also illustrated in the studies of Patient 0866 (Figs. 2*A* and 2*B*). The magnitude of the disproportion in the distribution of vital capacity (19 per cent) and residual volume (37 per cent) on the side of thoracoplasty would have been difficult to surmise in the latter without the unilateral figures. Although the very high proportion of the total lung capacity comprised by the residual volume in this patient's left lung (50 per cent) might be interpreted to indicate "overdistention," it is doubtful that this ratio has the same connotation in this instance that it usually carries where there is no deformation of the parietes and where there exists no gross alteration in the relationship between tissue mass and its enclosure.

It was observed in the description of results that the distribution of the residual volume varied more among the "normals" than did that of the other compartments. It should be noted that in the two instances in which departure from the mean was most pronounced (Patients 0586 and 0627), the residual volume on the side of the lesion was unusually low. A similar effect of relatively small, unilateral, parenchymal lesions on the ipsilateral residual volume was also found occasionally among patients selected for phrenic nerve interruption.[9]

One might anticipate that results with the bronchospirometric technique would be influenced by the resistance to respiration added by the Carlens tube.[10] Were this factor of major importance, the functional residual capacities measured by the unilateral technique should be higher than the bilateral figures.[11] Since such a difference exceeding 50 c.c. was found only once (Patient 0490, 286 c.c.), any additional resistance encountered by these 9 patients was probably not pronounced.

Early in the development of the unilateral technique, the concentration of nitrogen in "alveolar" air was frequently found to be abnormally high in comparison with the bilateral figure. In order to reduce the magnitude of this difference, the period of oxygen breathing for the unilateral procedure was extended to nine minutes as compared to the conventional seven. With this modification, concentrations below 2.5 per cent were found in thirteen of the eighteen unilateral analyses included in this report. Of the remaining five, one (Patient 0490, left lung) was associated with a high bilateral figure, two were observed in a patient with an unusually large functional residual capacity (No. 0612) and a marginal bilateral index (2.13 per cent), one could be attributed to poor ventilation (17 per cent of total) on the side of thoracoplasty (Patient 0866), and in the final instance (No. 0627, right lung) the elevation could be explained on no reasonable basis other than contamination. The likelihood of a continuous air leak in the latter is excluded by the normal distribution of the functional residual capacity and the close correspondence of unilateral and bilateral figures. A small admixture of room air during delivery of the sample or inadequate flushing of the alveolar air trap are most likely to have been responsible.

The basic reason for differences in nitrogen emptying rates with the two techniques, however, is in all likelihood our failure to reduce the volume of tubing and valves between inspiratory and expiratory orifices on the unilateral oxygen circuits commensurately with the reduction in tidal volume due to unilateral respiration. In this circumstance, the ratio of dead space to tidal exchange for the unilateral procedure is in excess of the figure for the conventional technique. Accordingly, a smaller proportion of the gases escaping through the expiratory valve are alveolar in composition, tidal volumes of oxygen are poured into a relatively larger reservoir during inspiration, and nitrogen elimination by serial dilutions is retarded.

Finally, it should be observed that the above considerations, although reflecting on the interpretation of the unilateral alveolar index, do not introduce appreciable limitations with respect to the comparison of unilateral and bilateral functional residual capacities. Because the equation for computing the functional residual capacity is relatively insensitive to final nitrogen concentrations, differences between unilateral and bilateral indices, unless extreme, result in small differences in the measurement of this volumetric compartment.*

SUMMARY

The residual volume of each lung individually was measured in association with bronchospirometry. In each of 9 patients, the sum of the functional residual capacities of the two lungs differed from the mean of two bilateral measurements by less than 10 per cent; in 8 of the 9 a similar level of agreement was found for the residual volume. The degree of correspondence for these volumetric compartments compared favorably with those established for the vital capacity and the total lung capacity.

Among 6 patients with minimal or moderate pulmonary tuberculosis and normal vital capacities the mean values for the distribution of the various compartments between the two lungs were nearly identical (54 per cent right, 46 per cent left for the functional residual capacity, vital capacity, and total lung

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*If the bilateral functional residual capacity were 3,000 c.c. and the mixing index 1 per cent, for example, a concentration as different as 5 per cent would result in a volume of 3,160 c.c. (error of 5.3 per cent).

capacity, and 55 per cent right, 45 per cent left for the residual volume). The extremes of variation around the mean were greatest with respect to the residual volume.

The advantage of knowing the unilateral residual volume was illustrated by application of the technique to 3 patients whose conventional bilateral measurements afforded an equivocal definition of volumetric status.

The bilateral studies and nitrogen analyses were performed by hospital corpsmen of the United States Navy trained in the Cardiopulmonary Laboratory of the U S. Naval Hospital, St. Albans, New York. The interest and skill of the following are acknowledged with particular gratitude: John Briggs, Kenneth Gorr, Roy Johannessen, Joseph Malin, Richard Monsees, Earnest Picknally, David Prior, and Robert Rauf

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MANAGEMENT OF THE POSTRESECTION SPACE IN TUBERCULOSIS

III. Role of Pre- and Postresection Thoracoplasty

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THORACOPLASTY, for several decades the major surgical treatment of tuberculosis, has seemingly receded into the limbo of the past since 1950.[1] With the rapid evolution of thoracic surgery and the spectacular use of anti-tuberculosis drugs, pulmonary resection has become the principal method of surgical treatment at the beginning of this fifty-year span.

Although the indications and results of collapse therapy have been clearly drawn, the majority of these operations were performed in the preantibiotic era.[2, 3] The successful application of various excisional techniques to all stages of tuberculosis has made it difficult to evaluate the role of thoracoplasty when associated with lung resection. Whether today's modification of the standard posterolateral thoracoplasty, the so-called apical tailoring thoracoplasty, is a useful adjunct in certain cases; or whether it is simply a carry over, and indeed should be discarded altogether, is not clear from current writing.

In accord with our study of the postresection space in tuberculosis, and various measures to avoid or treat such a space, we have reviewed the indications and results of pre- and postresection thoracoplasties, as performed at the Veterans Administration Hospital (Sunmount, New York), over a five-year period (1950 to 1955).

PRERESECTION THORACOPLASTY

During the period of transition (1940's), when thoracoplasty was gradually yielding to resection as the dominant surgical treatment, experienced thoracic surgeons believed that resection was safer for certain types of patients when they had had a previous thoracoplasty. Prior to the systematic application of prolonged, continuous chemotherapy, no methods other than major or minor collapse were available for the control of progressive, exudative disease. Patients with moderate or far-advanced tuberculosis, who showed no inherent resistance to their disease under conditions of maximum bed rest and improved nutrition, were treated with varieties of pulmonary collapse, often on an emergency basis.

The amazing success which specific antimicrobial agents have had in aiding the resolution of pneumonic or prenecrotic tuberculosis, has made the control of the acute case largely a medical, rather than surgical, problem. The average

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surgical candidate today may come to resection in the sputum-negative state, with bacilli on the last positive culture completely susceptible to the particular drugs being used.

A small group of patients, perhaps 10 per cent, even with the administration of several combinations of prolonged drug regimens, will not, after six to nine months, show permanent sputum conversion or cavity closure. In an effort to control these patients for ultimate resection, and following the doctrine of the thoracoplasty era, a number of preresection thoracoplasties were performed. Our specific indications were: patients with unequivocal upper lobe cavities and persistent or intermittently positive sputum in whom the greater part of the exudative element had cleared. Unfortunately, many of this group had bacilli resistant to one or two drugs and uncertain coverage with a third agent. The anticipation of pleural complications in this group, plus the desire to minimize them, led to a recommendation of preresection thoracoplasty. Further, it was hoped that a period of collapse might aid the stability of disease in these patients who had had only a moderate response to the drug program. A second consideration was more mechanical than chemotherapeutic. In this same group were patients with scattered middle and lower lung field lesions, associated with major upper lobe involvement. Fluoroscopy revealed a 2 to 3 plus trapping in the lower lobes, with various degrees of obstructive emphysema demonstrated in pulmonary function screening. The expectation was that such lower and middle lobes would exhibit delay in occupation of the apex following upper lobectomy or bisegmental excision. The prospect of a space problem was then a further reason for preresection thoracoplasty.

Our plan of preresection thoracoplasty has been the subperiosteal removal of the first 3 ribs, with a routine apicolysis down to or near the level of the azygos vein or aorta, respectively. This operation was carried out in 29 patients, without complications. The interval from thoracoplasty to resection varied from a minimum of three to eight weeks. In older patients, a longer interval was allowed for increased stability of the regenerating plate.

The extent to which a first stage, 3-rib thoracoplasty reduces over-all function is variable.[4] The physiologic age of the patient, the amount and distribution of tuberculosis in the underlying lung, the association of chronic bronchopulmonary conditions, such as asthma, emphysema, and interstitial fibrosis, are among the factors which make the functional loss unpredictable on an individual basis. It is generally assumed that a restricted collapse, over a relatively destroyed upper lobe, will result in very little net change.

Kaltreider and co-workers commented that thoracoplasty produces a greater reduction in the ventilatory capacity than is to be expected from the extent of lung tissue compressed. Cournand and associates,[6] in a study of 200 cases, found that an apical thoracoplasty (up to 5 ribs) decreased the maximal breathing effort up to 15 per cent of its preoperative value. In a similar study, Gaensler and co-workers[7] concluded that the maximal breathing capacity was decreased about 20 per cent when 5 or 6 ribs were removed. We have not carried out pulmonary function studies in patients who had a specific preresection thoracoplasty followed by resection. Since full physiologic recovery from thoracoplasty cannot be expected in less than four months (Gaensler found that loss of function at two weeks was nearly twice what it was at six months), it was felt that this determination would be of little value.

In order to avoid the physiologic handicap of paradox following thoracoplasty, it is customary to wait for variable intervals to allow hardening or bony regeneration of this area. Because the adequate clearing of secretions is dependent on the coordinate effort of all intercostal musculature with the diaphragm, "the bellows mechanism," even a rigid thoracoplasty plate means the upper third of the hemithorax is adding little to the cough mechanism. Pain from a fifth rib level incision further splints the lateral chest wall. In the case of a previous two-stage thoracoplasty, the plate itself may have to be cut for some distance when mobilizing the upper lobe for resection.

Analysis of the cough mechanism in human beings have shown that it is a complex coordination of muscular effort. Contraction of the intrinsic and extrinsic muscles of respiration, while the diaphragm aids the tussive force of the lungs in expelling secretions, indicates the importance of synchronous action and elastic recoil in the thoracic cage and its contents.

Consideration of the above observations, plus the impression of how a patient with a preresection thoracoplasty behaves after resection, compared with the behavior of a patient who has an upper lobectomy without thoracoplasty suggests that because of the presence of this thoracoplasty we have made these patients unnecessary surgical cripples.

Radiologic observations (Figs. 1, 2, and 3), during the first two postoperative weeks, reveal that the underlying lung is not ventilating as well, is not filling the hemithorax as promptly, nor clearing the operative reaction as soon as in the nonthoracoplasty patient. Particularly is the older patient handicapped. General muscle weakness, obesity, higher incidence of chronic emphysema and bronchitis, rob the man over 45 years of age of the extra respiratory effort he needs so badly.

There have been no tuberculous complications incident to the preresection thoracoplasty itself. Because it is a safe procedure, of course, does not mean it is necessarily a wise one. Major complications have followed the resection itself, and are probably more related to the chemotherapeutic control and the presence of unsuspected endobronchial disease and resistant organisms. There is no evidence to suggest that any of these complications were in any way avoided; in fact, some may have been precipitated by the presence of a preresection or standard thoracoplasty.

Minor postresection "mechanical" complications are, however, definitely increased in the preresection thoracoplasty group. Almost without exception, they have had a more difficult convalescence. Intratracheal aspiration, bronchoscopy, intermittent oxygen, aerosol mixtures and bronchodilators are all required with greater frequency in these patients.

There is no way to reconcile the hazards of performing a thoracoplasty simultaneously with a resection, unless paradox can be eliminated either by an

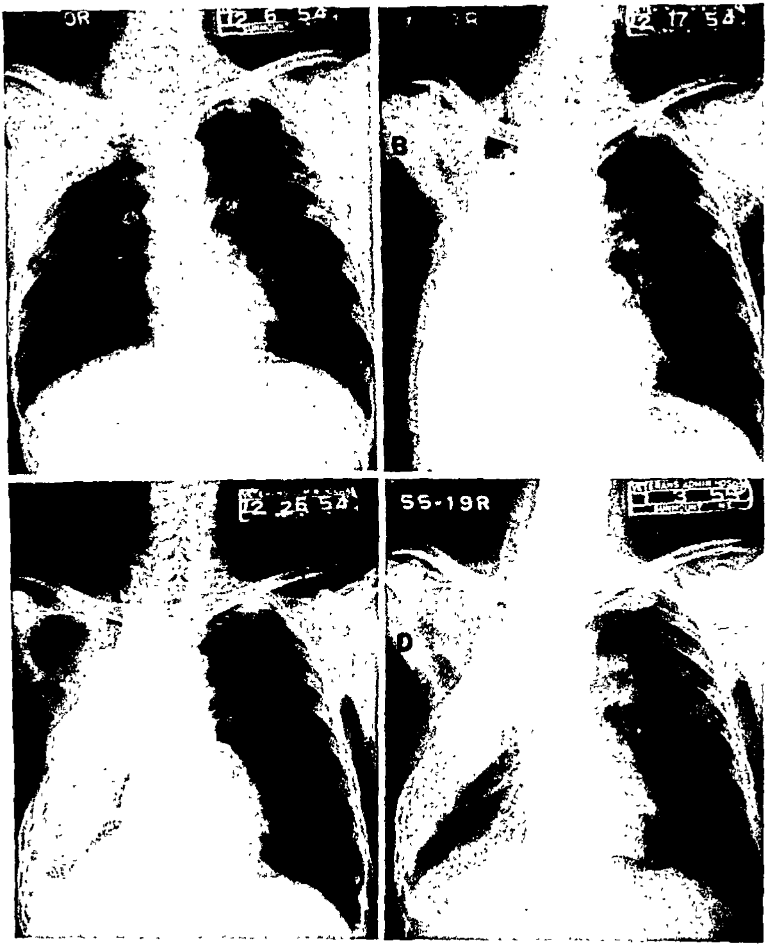

Fig. 1.—*A*, Right preresection thoracoplasty, one week before resection. *B*, four days following right upper lobectomy. *C*, Thirteen days postoperation. *D*, Twenty-one days postoperation.

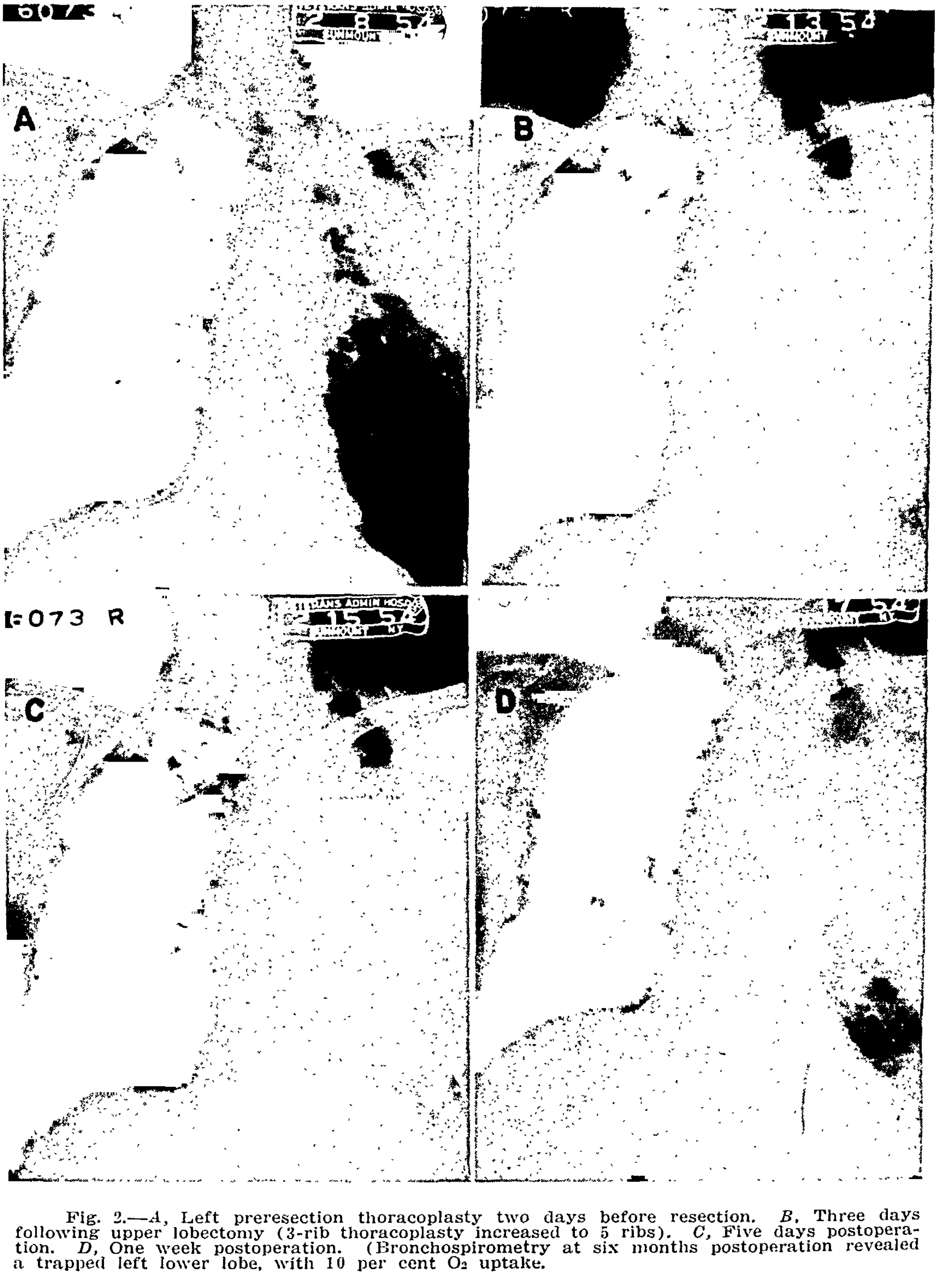

Fig. 2.—*A*, Left preresection thoracoplasty two days before resection. *B*, Three days following upper lobectomy (3-rib thoracoplasty increased to 5 ribs). *C*, Five days postoperation. *D*, One week postoperation. (Bronchospirometry at six months postoperation revealed a trapped left lower lobe, with 10 per cent O_2 uptake.

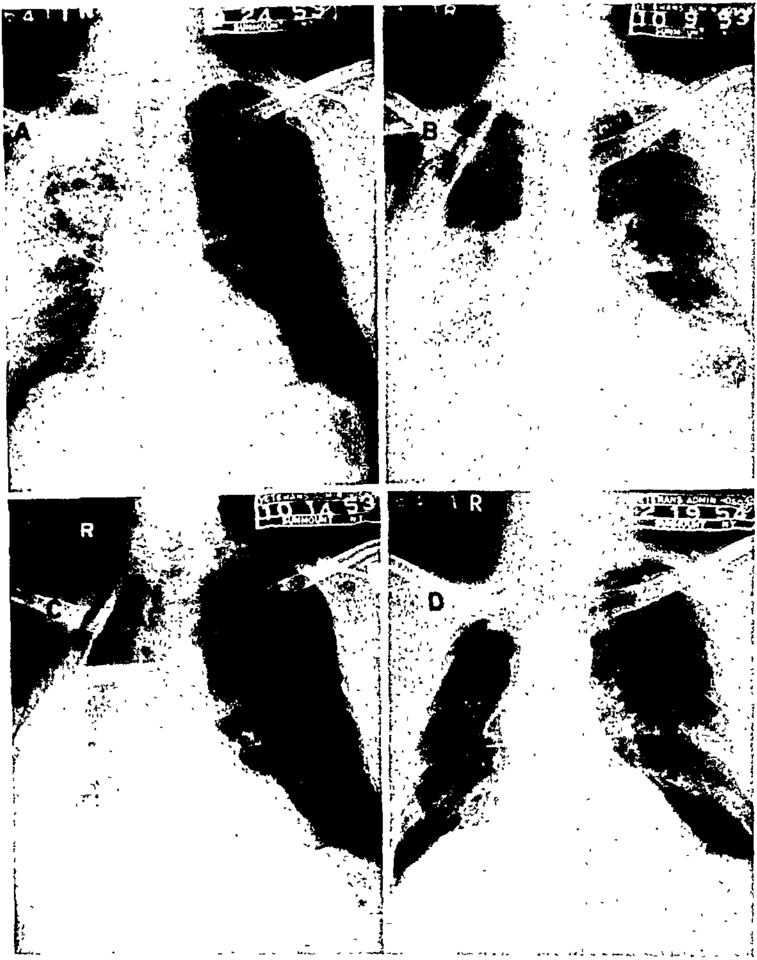

Fig. 3.—*A*, One day following right preresection thoracoplasty. *B*, One day following right upper lobectomy. Note communication with subscapular space. *D*, Six days postoperation. *E*, Four months postoperation. Small residual apical space.

extraperiosteal plombage or osteoplastic procedure of Björk.[8] Since the mechanical disadvantage to the patient is essentially the sum total of both procedures, it is only the young patient with good reserve who can stand this combined insult.

In 11 patients receiving concomitant thoracoplasty following upper lobectomy, Sze, Samadi, and Conant[9] reported postoperative atelectasis in 7. Similarly, J. K. B. Waddington[10] recorded varying degrees of ipsilateral atelectasis in 9 of 20 patients following thoracoplasty concomitantly with resection.

PLEURAL TENTS

Fashioning of a cap or tent of parietal pleura to lower the apex of the chest, and aid in covering raw lung surfaces, has been a valuable adjunct.

The technique of pleural tenting has been described by Miscall and Duffy.[11] It is similar to the creation of an extrapleural pneumothorax, with the exception that it is much easier to perform from within the thorax.

While small rents in the pleura may be sutured, any extensive defect incurred from entry into the extrapleural plane, during mobilization of the upper lobe, makes formation of an intact tent difficult or impossible.

It is possible to tailor the pleural dissection in such a manner that it may be posterolateral, apical, or anterolateral, and overlie the area of resected lung and maximum raw surface. Construction of such a tent adds only 15 minutes to the resection time. The effectiveness of this temporary roof may be estimated before the chest wall is closed.

No effort is made to maintain an extrapleural space with air or liquid during the postoperative period. The space is quickly filled with blood if the pleura remains intact, and shrinks gradually during the successive two or three postoperative months (Fig. 4). In a few cases, however, an apical air pocket (extrapleural) has persisted, and acting on the observation that such a space might indeed be preventing postoperative expansion of the residual lung, we have carried out transient aspiration with needle or catheter.

Our over-all results in this group (25 cases) have been most satisfactory. Whenever it has been used, the pleural tent has been effective in minimizing a potential postresection space and has assisted in reducing the incidence of transient air leakage from the raw surface of lungs with limited pneumatic (self-sealing) function.

POSTRESECTION THORACOPLASTY

A commonly accepted indication for postresection thoracoplasty in tuberculosis surgery has been a persistent apical space unfilled by remaining lung. Although the existence of such a space is usually, but not always, associated with a bronchopleural fistula,[12] the adjunctive "space-filling" thoracoplasty has often been successful in the dual role of space obliteration as well as fistula closure.

Sze, Samadi, and Conant,[9] in a review of their resection group, formerly used a 3- or 4-rib thoracoplasty routinely, following upper lobectomy, ostensibly to prevent overexpansion of the remaining lung and reactivation of residual

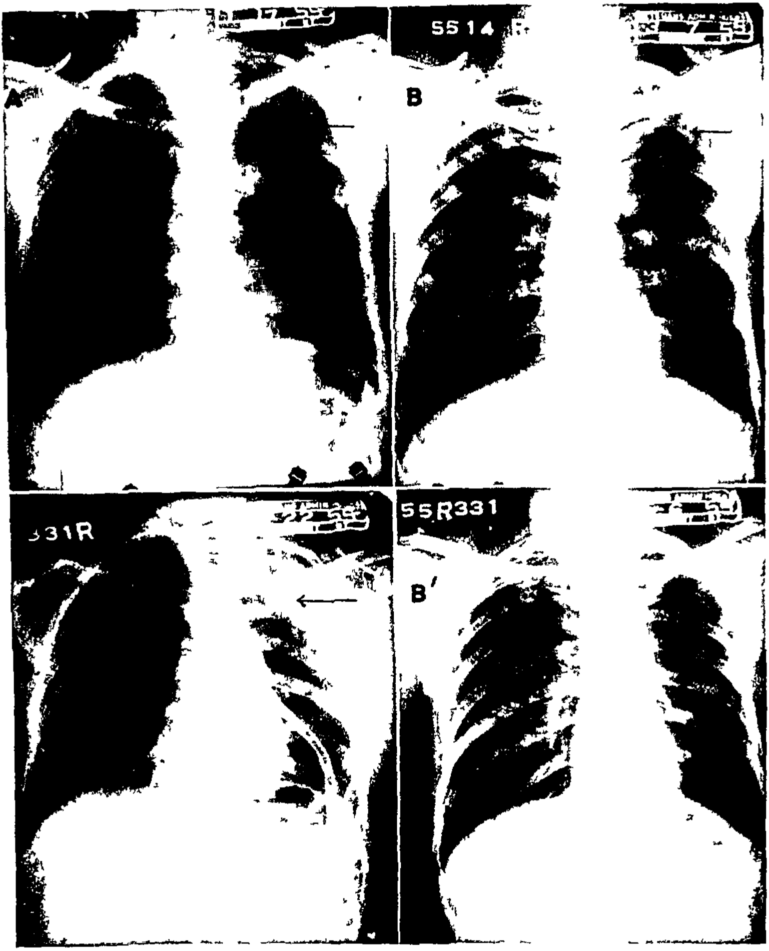

Fig 4.—Pleural tents *A* Twelve days following excision of superior division left upper lobe *B* Two months postoperation Lung at level of 5th rib posteriorly *A′* Two days after left upper lobectomy Tent at level of resected rib *B′*, Five and one half months after left upper lobectomy Pleural tent has disappeared Left lower lobe filling hemithorax.

disease. At present, with the protection afforded by postoperative chemotherapy for six months or longer, they reserve the postresection thoracoplasty for the occasional patient with residual pleural space or bronchopleural fistula.

Murphy[13] reserved the subsequent thoracoplasty for the case with incipient or frank bronchopleural fistula, or persistent delay in re-expansion. Following this policy, he obtained 82.7 per cent satisfactory results after 98 operations (segmental resections) without a thoracoplasty.

Our indications for the postresection thoracoplasty have been discussed previously[14] and, in general, are similar to those of Murphy.

The need for a small "space obliterating" apical thoracoplasty, subsequent to resection, appears to decrease proportionately as the mechanical problem of dealing with the cut surface of the lung is solved.

The incidence of early or late tuberculous complications in the patient receiving long-term drug therapy, and coming to surgery with susceptible bacilli is quite small. We believe that the indications for a postresection thoracoplasty should now include the bacteriologic consideration of the patient's sputum preoperatively, the presence of resistant bacilli in the sputum or resected material, and the distribution, character, and amount of residual parenchymal disease.

Recently we have been attempting surgical treatment in a group of patients with cavitary upper lobes, positive or intermittently positive sputum, and resistant bacilli. In this group (now numbering 12 patients), we have been aware of the impossibility of removing all palpable disease by preoperative laminography.

The cavity-bearing upper lobe has been removed, leaving residual closed lesions (caseous, necrotic foci) in the lower lobe and contralateral upper lobe, up to, and in some cases, exceeding 2 cm. While the lower lobe may undergo some compensatory emphysema (or already has obstructive emphysema), the contralateral upper lobe may do the same with shifting of the superior mediastinum. In this group with resistant bacilli in the resected specimen, and usually in the regional lymph nodes, we have advised postresection thoracoplasty, largely on the basis of poor chemotherapeutic control rather than the fear of overdistention leading to subsequent reactivation.

DISCUSSION

Björk[15] lists the disadvantages of a preliminary thoracoplasty with secondary resection as follows:

1. Psychological handicap of facing a second major operation.
2. Prolonged hospitalization.
3. The danger of spread is greater after a thoracoplasty than after a resection.
4. It is more difficult to model the chest in the desired size at a second operation.

The physiologic evidence of the handicap which a preresection or concomitant thoracoplasty imposes on a patient is considerable. Björk[16] observed several instances where 5 rib thoracoplasties with apicolysis resulted in more severe postoperative courses than resection in similar cases He believed this was due to the combination of compression atelectasis, produced by the thoracoplasty, and the accumulation of fluid in the apicolysis space, plus paradoxical movements of the unstable chest wall In a series of 16 patients, following resection of segments and lobes under old thoracoplasties, he found that in patients with good pulmonary function, the arterial O_2 tension reached the preoperative value on the fifth day after surgery, whereas in those with advanced bilateral disease, the arterial O_2 tension had not reached the preoperative values after fourteen postoperative days

W W Stead and co-worker,[1] [18] in a series of valuable physiologic studies following thoracic surgery, confirmed the work of Bjork and Berggren[17] in finding a fall of hemoglobin saturation by a shunt of blood through recently collapsed lung tissue which was not being aerated They found, as had other investigators, the existence of a definite respiratory acidosis at the completion of the operative procedure They determined that in order to correct this acidosis, an increase in alveolar ventilation was required In the patient who was unable to effect this increase in minute ventilation, because the tidal exchange was limited by pain and varying degrees of paradox, the compensation was met by the more inefficient mechanism of a marked increase in the respiratory rate In patients who were unable to meet this demand of extra respiratory work because of age, debility, extent of surgery, or associated pathology, which had already compromised their reserve, serious degrees of pulmonary insufficiency or death might result in the postoperative period

Bjork considers the only advantage of staging the thoracoplasty and resection to be the relative absence of paradox at the second procedure In view of the manifest psychological, surgical, and physiologic disadvantages, there must be more than the lack of paradox to justify the widespread impression that a preliminary thoracoplasty makes resection more safe for a patient[19] Are there certain patients in whom the drugs are unable to effect the necessary resolution of disease, so that the patient is a better risk for resection? Should a collapse procedure be carried out on these patients with the anticipation of failure therefrom, and the deliberate intent to resect later in any event?

Since a satisfactory thoracoplasty (Alexander or plombage) may arrest up to 80 per cent of cases, it is difficult for us to determine which will be the more unfortunate 20 per cent, without allowing the test of time If preresection thoracoplasties are done as a routine measure in the open positive group, will not a considerable number of patients have resections whose disease might well have become arrested with the thoracoplasty alone?

In 1952, Takaro, Clagett, Hodgson, and Carr,[0] in reporting 73 cases of pulmonary resection for tuberculosis, with a follow up of one year or longer, stated that "it is generally agreed now that thoracoplasty should be performed after pneumonectomy or lobectomy for tuberculosis, to prevent overexpansion of the remaining pulmonary tissue and breakdown of quiescent tuberculous foci." This dogma, echoed by many writers in the ensuing years, has become, possibly through strength of repetition, a traditional indication for a concomitant or postresection thoracoplasty.

There are no adequate physiologic or pathologic data to support the contention that true emphysema develops in the remaining lung tissue following lobectomy. Experimental studies,[21, 22] which are largely based on pneumonectomies in dogs, reveal the presence of simple distention, and, later, compensatory emphysema. Whether these changes progress to emphysema in the anatomic sense, with atrophy and rupture of alveolar septa, following *upper lobectomy* in young patients, has not been conclusively demonstrated. Nor is there any good evidence that this amount of lower lobe hyperinflation is a physiologic deficit, or, for that matter, can necessarily be prevented by a postlobectomy thoracoplasty.[22-24] Various degrees of obstructive or localized hypertrophic emphysema already exist in these lungs at the time of resection, apparently as a result of the healing processes of tuberculosis.

If the emphysema-preventing thoracoplasty is to be advocated on a prophylactic basis, it would seem that substantial evidence should be available that such lower lobe distention is a physiologic handicap, and, moreover, that such late changes may be prevented by thoracoplasty.

There have been sporadic case reports of tuberculosis beginning in upper lobes following lower lobectomy for bronchiectasis.[25-27] In almost every reported case, there was no evidence of tuberculosis on the pre- or immediate postoperative chest films. Progressive tuberculosis appeared in the majority of these cases within one year after lobectomy. Rothstein and Gerson[25] attribute this activation of tuberculosis to overexpansion of the upper lobe. However, none of the authors refer to their gross findings at operation, and all cases were done without the benefit of any specific antituberculosis chemotherapy.

We should like to emphasize the extreme unpredictability of the residual necrotic focus. Reactivation of tuberculosis has occurred under a variety of conditions. There is no information in the previous reports which establishes a cause-and-effect relationship of overdistention on small residual lesions in the remaining lobe.

In addition, we have seen 2 patients who had a postlobectomy thoracoplasty and were treated for six months postoperatively, yet had lower lobe reactivation three years later.

In this regard we accept Medlar's[28] concept of pathogenesis; i.e., the spread of tuberculosis is due to bronchogenic dissemination. He has furnished pathologic evidence which reveals that, while the healing process takes place to a certain extent in a collapsed lung, the necrotic lesion is irreversible and maintains its proximity to the bronchus. While actual sloughing of the parenchymal lesions may be prevented by certain mechanical conditions, true healing is rarely seen. He found, in 1 case, a chronic, sloughing, open cavity, with bronchial communication, which had been under a thoracoplasty for fifteen years. Dr. Medlar[29] states further, he has never seen a specimen in which reactivation from a latent caseous lesion was conclusively due to the process of overexpansion.

In conclusion, it would seem that if adjunctive thoracoplasty is to prove its value in association with resection surgery for tuberculosis, there must be definite evidence of protection against major tuberculous complications in the thoracoplasty group Complications (fistula, empyema, spread) following lobectomy (411 cases), with and without previous thoracoplasty, as reported in one year of tabulated resection data from 47 contributing hospitals (14th VA, Army Navy Conference on Chemotherapy of Tuberculosis)[30] do not indicate that any protection is afforded by the preresection thoracoplasty Overholt, Wilson, and Gehring[31] concluded in 1952, that, Chemotherapy has produced a profound reduction in the risk associated with these operations Thoracoplasty done before, with or immediately after resection did not lower the complication rate in our series ” If this statement was true in 1952, it would appear even more justified in 1955

SUMMARY

1 A series of 29 cases of preresection thoracoplasty, 25 cases of pleural tent, and 25 of postresection thoracoplasty is presented with their clinical indications

2 The physiologic handicap of the preresection and concomitant thoracoplasty is emphasized

3 There is no definite pathologic or clinical evidence that the adjunctive thoracoplasty makes resection more safe for a patient

4 The impression that reduction in the size of the hemithorax should benefit patients with obstructive or compensatory emphysema is not tenable These are the patients who are most vulnerable to any interference with the bellows mechanism of thorax and diaphragm

5 Prophylactic thoracoplasty, for the sake of preventing overdistention or reactivation of tuberculosis is a nebulous concept A thoracoplasty for the purpose of closing a fistula or empyema space is therapeutic

6 The foregoing statements are not presented with any intent to *settle an issue* The plea is made that, if a patient is to be given an adjunctive thoracoplasty, there must be considerably better testimony than is now available that it is worth the price he pays

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THE PERSISTENT PLEURAL SPACE FOLLOWING PARTIAL PULMONARY RESECTION

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INTRODUCTION

ALTHOUGH the goal in the postoperative management of patients having partial pulmonary resection is to re-expand the lung rapidly and bring it into apposition with the chest wall, there are a certain number of patients where this is not accomplished. A persistent pleural air space is not infrequently an x-ray finding after partial pulmonary resection. This finding is a cause of concern for the surgeon. The hazard of infection, especially suppuration with attendant extension into other parts of the lung or pleural space, prompts the surgeon to obliterate the space by early vigorous measures. These include needling and aspiration, closed drainage with or without suction, and, very often, thoracoplasty.

Now, however, with the use of antibiotics many complicating infections may be prevented. This study is a review of data on patients who showed a persistent pleural air space in order to observe the fate of the space with and without secondary surgery.

PATHOGENESIS

An early postoperative space may be due to a disproportion in volume between the chest cavity and the remaining lung tissue. Without an air source, this type of space gradually contracts, fills in with exudate, and obliterates. A pleural space which persists is strong evidence of bronchial air leakage. If the air source is large as from a lobar or segmental bronchus, this fistula results in an infected space. However, if the air source is small as from a terminal or respiratory bronchiole, and the patient is under antibiotic treatment, there is a high probability that the patient will be symptomless and the space would go unrecognized but for the finding on chest roentgenogram.

METHOD

All patients undergoing partial pulmonary resection at Triboro Hospital between July 1, 1951, and June 30, 1953, were studied. Selected preoperative x-rays were scanned for the type of disease, and all postoperative x-rays of each patient were serially reviewed to determine whether there was a pleural

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space. When a space was present twenty-one or more days after operation, it was recorded as a persistent space. Subsequent films were examined to determine the fate of the space and how long it persisted. At the same time the clinical record was investigated in detail.

These patients were nontuberculous and tuberculous. In the tuberculous patients, if organisms were found in the sputum or gastric contents within ninety days before the day of surgery, the patient was classified as sputum positive. The remaining are tabulated sputum negative.

Preoperatively, all tuberculous patients received combined long-term chemotherapy until maximum benefit was obtained, determined by chest roentgenogram and sputum studies. Postoperatively, drug therapy was continued for at least four months. The nontuberculous patients received suitable chemotherapy before and after surgery. In all cases, the decision for surgery was made at a medical-surgical staff conference.

Postoperatively, the patient's course was noted for fever, tachycardia, respiratory symptoms—especially cough, hemoptysis, wheeze, sputum both as to volume and character—and body weight. The persistence and amount of air leakage through the chest drainage tubes as well as the character of the fluid drainage were observed. Supplementary surgical procedures were tabulated, and in tuberculous patients the sputum was examined and cultured for bacilli. These observations extended from the time of surgery to the last follow-up visit. This represents a follow-up from one and one-half to three and one-half years.

PRESENTATION OF DATA

This study comprises 136 patients (Table I) who had partial pulmonary resection. There were 104 procedures for pulmonary tuberculosis, of which 33 cases were sputum positive. There were 32 operations for nontuberculous pulmonary disease.

TABLE I. TYPES OF PARTIAL PULMONARY RESECTION IN 136 PATIENTS

	TUBERCULOUS		NON-TUBERCULOUS	COMBINED TOTAL
	SPUTUM			
	POSITIVE	NEGATIVE		
Lobectomy	14	25	20	59
Segmental	12	33	5	50
Wedge	0	1	4	5
Lobectomy and Segment or Wedge	7	12	3	22
Total	33	71	32	136

After study of all postoperative roentgen chest films on these patients, which included tomograms in the anterior-posterior and lateral projections, 41 were found to have a pleural space twenty-one or more days after lung resection. Without the routine use of sectional radiography, the identification of many of these pleural spaces would have been difficult, particularly so in determining a space under a thoracoplasty. Postoperative fibrotic strands and emphysematous lung and blebs have been hard to distinguish from a space without this aid A considerable number of these spaces would not have been detected on conventional posterior anterior and lateral films

The pleural air spaces were evident generally from the first day following lung resection and could be followed throughout their course Several, however, appeared a week or more after surgery, and some after removal of the chest drainage tubes

Postoperative spaces were followed on serial roentgenograms and the time of their disappearance recorded Such patients have been followed postoperatively for a period of from one and one half to three and one half years All are still being followed except for two who did not return to the clinic after disappearance of the space on x-ray

Clinical records of the 41 patients with pleural spaces were examined in detail After operation, 14 of these had persistent symptoms such as cough, wheeze sputum production, fever, tachycardia, and weight loss These patients have been classified arbitrarily as having bronchopleural fistulas The remaining 27 asymptomatic patients make up the group of particular interest in this study They had an uneventful postoperative recovery and on clinical grounds could be considered uncomplicated The presence of a space on x ray was not anticipated

ASYMPTOMATIC GROUP—PERSISTENT PLEURAL SPACE

Table II classifies these asymptomatic patients according to type of resection and Fig 1 shows the type of disease which they had at the time of pulmonary resection There were 20 operations for pulmonary tuberculosis Six of these patients had positive sputum The remaining seven operations were for nontuberculous pulmonary disease It will be seen that the incidence of spaces in segmental and wedge resections, or combinations of these, exceeds that from lobectomy This is because the parenchymal dissection inherent in the procedure involves a greater surgical surface of lung tissue from which air leakage occurs

TABLE II INCIDENCE OF ASYMPTOMATIC PLEURAL AIR SPACES IN 136 PATIENTS ACCORDING TO TYPE OF PULMONARY RESECTION

	TUBERCULOUS SPUTUM		NON TUBERCULOUS	TOTAL	PERCENTAGE WITH SPACES (PER CENT)
	POSITIVE	NEGATIVE			
Lobectomy	0	4	2	6	10 2
Segmental	3	6	2	11	22 0
Wedge	0	0	3	3	60 0
Lobectomy and Segment or Wedge	3	4	0	7	33 2

Fig 2 shows how long the spaces persisted on tomographic roentgenogram One half of them had disappeared before five months There were seven cases which were followed for a period of over seven months (Fig 3) Three had spaces which were persistent after two years All these patients are on full activity and remain free of symptoms. One tuberculous patient was found on follow-up, ten months after discharge, to have a cavity in the contralateral lung. The pleural space had been closed for six months, however, and the ipsolateral lung parenchyma remained free of disease. The sputum had also been negative during this time.

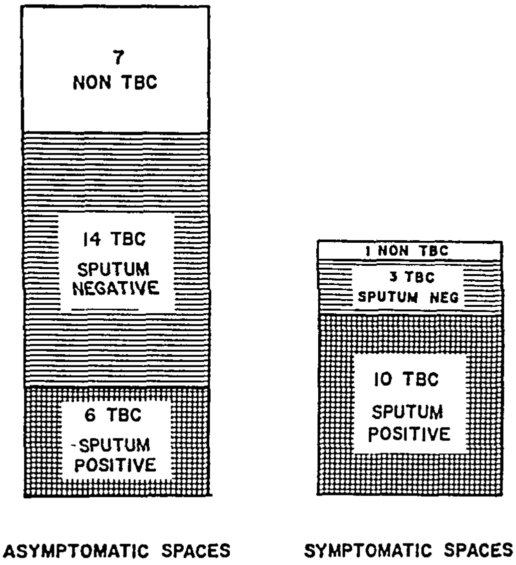

Fig. 1.—Type of disease in patients having spaces.

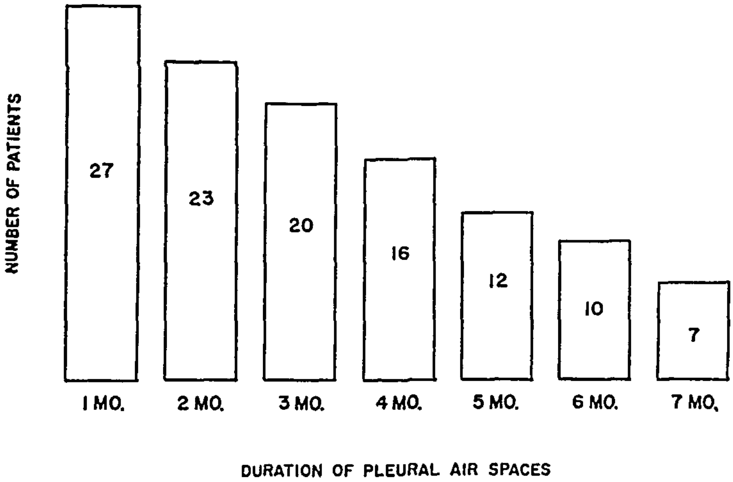

Fig. 2.—Duration of the pleural air space in 27 asymptomatic patients. Three patients have shown a persistent pleural space for over two years.

In reviewing the surgical management of this group without symptoms, it was found that 20 patients received no supplementary surgery although at conference the advisability of thoracoplasty was considered regularly. One patient had a limited thoracoplasty at the time of resection. Several patients refused further surgery because of their well-being. There were seven who had operative intervention. One had catheter drainage for twenty-four days.

Six had surgical exploration for the pleural space and limited rib resection over the space. Three of the patients in whom thoracoplasty was carried out continued to have the space for one year or more after the secondary procedure.

Exploration of the spaces in the 6 asymptomatic patients, just mentioned, revealed findings which were quite uniform. Their size varied from 3 to 10 cm. They were located in the upper half of the chest between the operated surface of the lung and the chest wall. During the first four to six weeks, the space was covered with exudate and organizing tissue. Minute openings could be demonstrated on the surgical lung surface from which air leaked with positive endotracheal pressure. Those spaces opened six months or more after resection were found to be endothelized and had a cystlike appearance (Fig. 4). The same minute bronchiolar openings could be demonstrated. None of these asymptomatic spaces explored was found to contain fluid or evidence of infection. Cultures for pyogenic organisms and tubercle bacilli did not show growth.

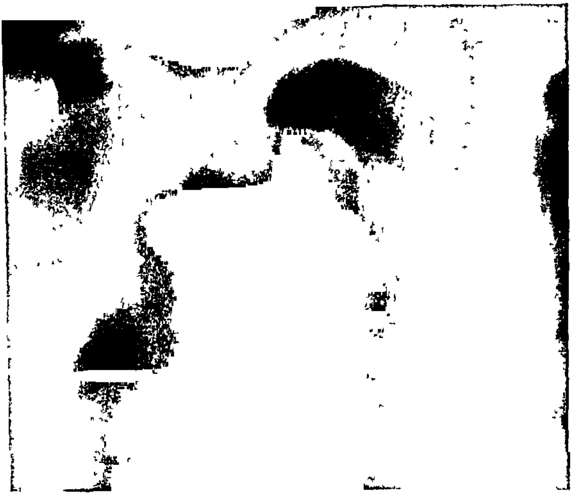

Fig. 3.—Tomogram of asymptomatic apical pleural space three months after resection of apical and posterior segments for bullous cysts. After ten months of observation it disappeared to x-ray view.

SYMPTOMATIC GROUP—BRONCHOPLEURAL FISTULA

Table III shows the distribution according to the type of resection of 14 patients with symptoms of bronchopleural fistula and Fig. 1 shows the type of disease they had at the time of pulmonary resection. There were 13 operations for pulmonary tuberculosis, ten of which were in patients who had tubercle bacill

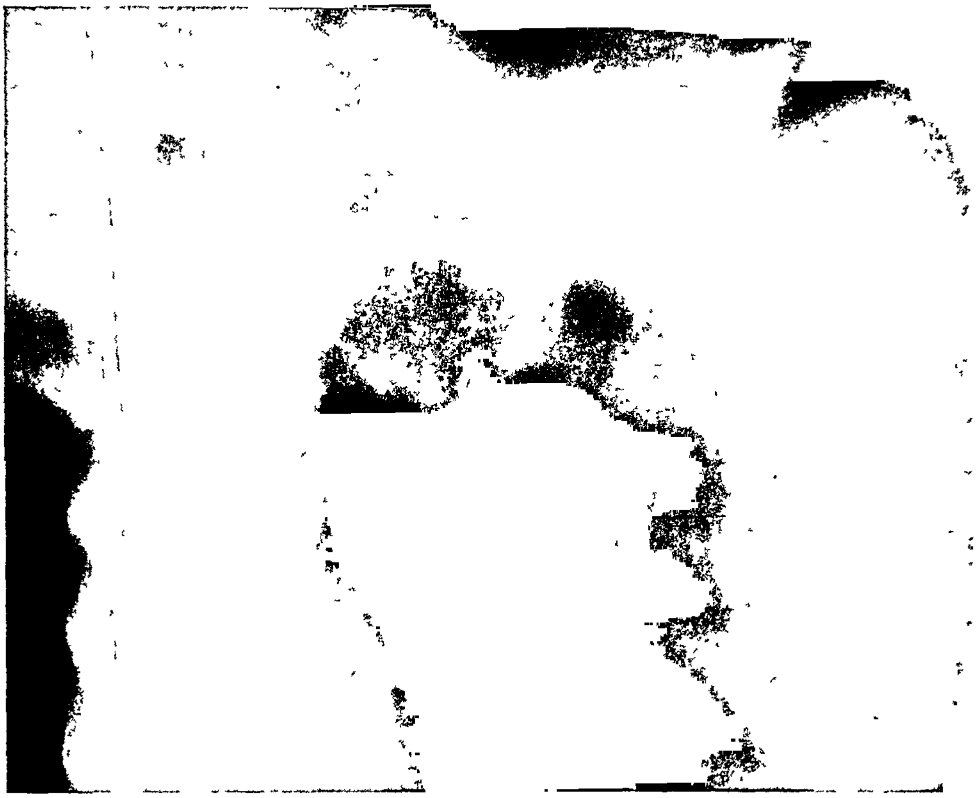

Fig. 4.—Tomogram of asymptomatic apical pleural space which was explored eleven months after resection of the left apical posterior segment for cavitary tuberculous lesion. The space was found epithelized and cystlike in appearance.

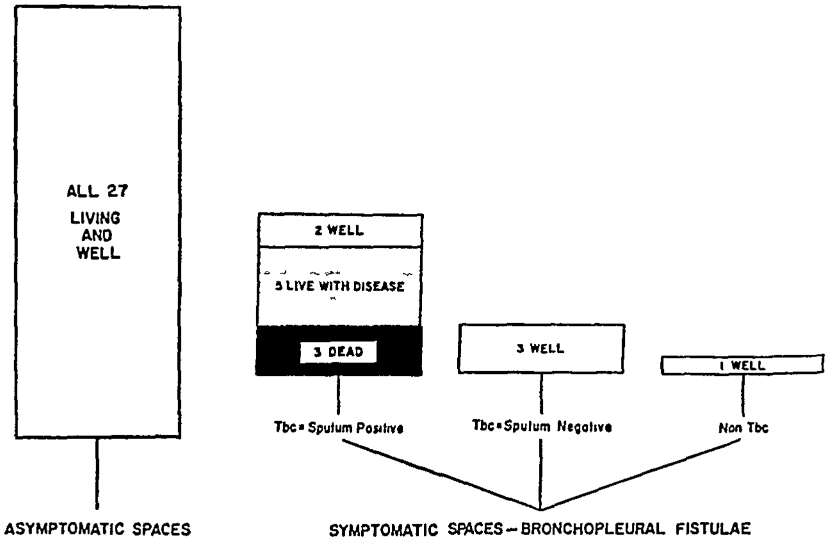

Fig. 5.—Present status of patients with asymptomatic and symptomatic spaces followed for one and one-half to three and one-half years.

TABLE III. INCIDENCE OF SYMPTOMATIC PLEURAL SPACES (BRONCHOPLEURAL FISTULAS) IN 136 PATIENTS ACCORDING TO TYPE OF PULMONARY RESECTION

	TUBERCULOUS SPUTUM		NONTUBERCULOUS	TOTAL	PERCENTAGE WITH FISTULAS (PER CENT)
	POSITIVE	NEGATIVE			
Lobectomy	5	0	1	6	10.2
Segmental	1	2	0	6	12.0
Wedge	0	0	0	0	0.0
Lobectomy and Segment or Wedge	1	1	0	2	9.0

middle lobe. There is in our group an approximate 10 per cent incidence of bronchopleural fistula, the incidence being similar for segmental resection and lobectomy. All these patients have had prompt chest drainage, multiple stage thoracoplasty, and various plastic procedures. Some have had further pulmonary resection and others, early secondary closure of an open bronchus.

Despite aggressive surgical attack, it will be noted in Fig. 5 that after a follow-up from one and one-half to three and one-half years on the ten patients with positive sputum that three are dead, four having persistent fistula live with disease in the hospital, and 1 lives at home. In the remaining two, the fistulas have closed and the patients appear well.

The three tuberculous patients with negative sputum who had bronchopleural fistulas are free of symptoms and fully active. Two of these continue to have apical spaces under a thoracoplasty. The remaining one, two months after upper lobectomy, had a secondary closure of an open bronchus with complementary thoracoplasty which obliterated the space. She also is well.

One nontuberculous patient with a bronchopleural fistula healed after open drainage and thoracoplasty.

DISCUSSION

We are impressed with the uneventful recovery of those asymptomatic patients with negative sputum who show on x-ray a persistent pleural space after partial pulmonary resection. None of these patients to the present time has developed complications because of the space. Prior to the antibiotic age, infection would be anticipated and hence obliteration of the space by operative intervention strongly advocated. The need for this additional surgery as a routine may now require evaluation, for the majority of these spaces will obliterate in a few months, and there appears to be little risk in following them for a year or more. A decrease in size of the space on serial roentgenograms is a favorable sign.

On the other hand, a patient with a pleural space whose sputum continues positive for organisms, in spite of drug therapy, has an uncertain prognosis for recovery. All deaths and serious morbidity in our series occurred among patients infected with antimicrobial resistant organisms (Fig. 5).

SUMMARY

The postoperative x-rays of 136 consecutive patients undergoing partial pulmonary resection were scrutinized for a pleural air space present twenty-one or more days after operation.

There were 41 patients in whom a persistent pleural air space was found and the significant clinical data were correlated.

Soon after operation, 14 of these patients had respiratory and constitutional symptoms indicative of bronchopleural fistula, ten of whom were infected with drug resistant tubercle bacilli. For the latter group, the prognosis was poor despite multiple operations; three are dead, five live with disease, and only two are clinically well. The remaining four patients whose infective organisms were preoperatively controlled by chemotherapy are clinically well after supplementary surgery.

There were 27 patients with pleural air spaces who were asymptomatic. They have had a satisfactory recovery with or without supplementary surgery.

This study shows that a postoperative pleural air space may be safely treated conservatively if the patient is carefully observed and the following conditions obtain:

1. There must be absence of respiratory and constitutional symptoms.
2. There should be progressive shrinkage of the space on serial roentgenograms. Tomograms are very helpful in this follow-up.
3. In tuberculous patients, the sputum must be negative for tubercle bacilli.
4. The patient should be on effective antibiotic drug therapy for control of the parenchymal disease.

If these criteria are not met, the patient should have early surgical intervention to obliterate the space.

THE MANAGEMENT OF THE PLEURAL SPACE FOLLOWING PARTIAL RESECTION OF THE LUNG (ESPECIALLY FOR TUBERCULOSIS)

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INTRODUCTION

THE obliteration of the pleural cavity following partial removal of a lung is most important. Alert surgeons strive for this ideal as early as possible in the postoperative period. A pleural space that has persisted for more than two weeks may lead to chronic empyema with or without bronchopleural fistula. Such complications often require further deforming and sometimes difficult operations, and the convalescence of the patient is prolonged. With eventual healing, fibrosis of pleura and diaphragmatic deformity may lessen future pulmonary function of the operated lung.

At Fitzsimons Army Hospital, over four hundred lobectomies or partial lobectomies were done during the past two years. Two thirds of these involved the resection of lobar segments that was performed usually for tuberculosis. It is very important that the postoperative tuberculous patient recovers without a pleural space or bronchopleural fistula. Such residuals usually prevent ultimate cures of the disease and lead to difficult problems in total management. It has been noted, however, that this group has recalcitrant pleural difficulties which tax the ingenuity of the surgeon. The Thoracic Surgical Service at Fitzsimons Army Hospital for the past two years has included students of this specialty from different parts of the country. They have contributed to the evolution of management of the pathology in question. Considerations for the care and prevention of pleural spaces after subtotal pulmonary rsection follow.

THE ADJUVANTS AND CAREFULNESS OF SURGERY AT THE TIME OF RESECTION

The type of operation and the amount of lung to be removed should be planned as completely as possible before surgery. Ideally, the lung remaining after resection should be free of inflammatory and mechanical disease. A bronchiectatic remnant does not expand well postoperatively and it is prone to develop areas of pneumonitis. The suturing of a divided bronchus with a tuberculous ulcer may result in a fistula later. These factors and many others must be diagnosed beforehand so that proper surgery may be selected.

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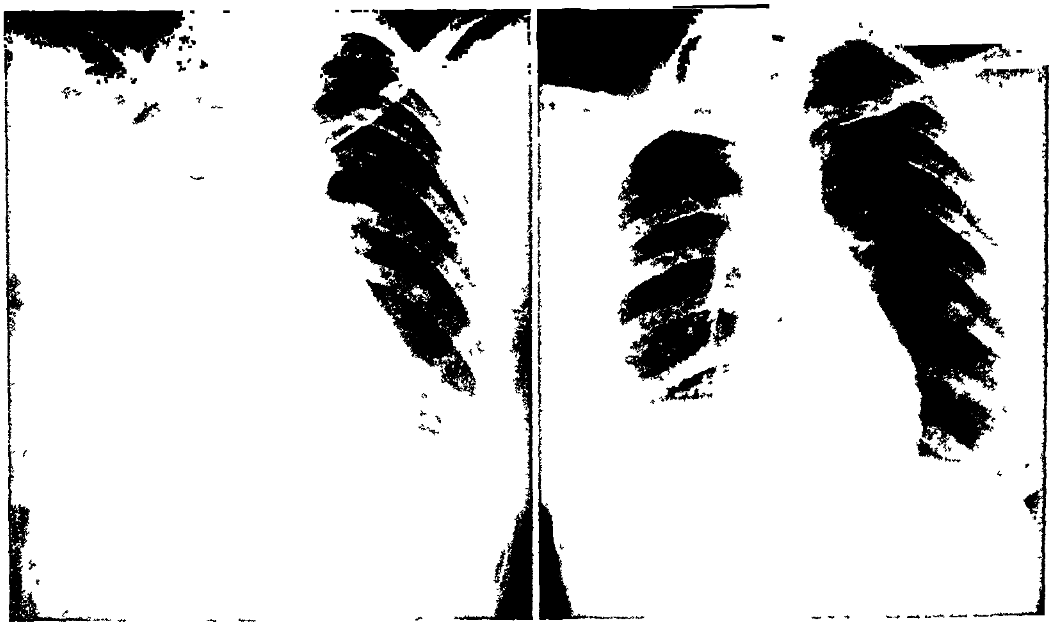
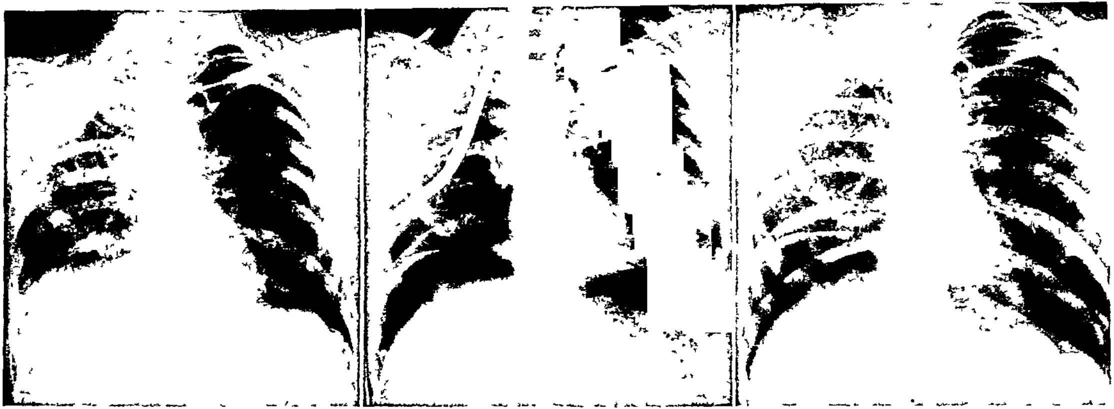
Preliminary thoracoplasty in tuberculous patients is a subject of much debate. Some surgeons prefer to use it at the time of resection or as a later procedure. At Fitzsimons, a modified thoracoplasty is done about three weeks prior to resection if the removal of more than two segments of an upper lobe is contemplated. The posterior half of the third rib and all of the second rib are resected subperiosteally leaving the transverse processes intact. The periosteum is stripped from the undersurface of the first rib and an apicolysis is performed down to the level of the third or fourth rib posteriorly (Fig. 1, A and B). If three or more segments are excised without this preliminary step, the modified thoracoplasty is done several weeks later. Concomitant procedures are not done unless the equivalent of four or more segments is removed and a first-stage thoracoplasty already has been done. Under such conditions a second stage may be completed at the time of resection. An example would be the excision of an upper lobe and the superior segment of the lower lobe in a patient that had a preliminary thoracoplasty. The fifth rib would be removed for the intrathoracic procedure and a posterior segment of the fourth rib would be removed near the end of the operation (Fig. 2, *A*). There is minimal paradoxical motion following this method of management. The relaxed chest wall approaches the small lung remnant and often leads to early obliteration of the pleural cavity. It also prevents overexpansion of the underlying lung.



A. *B*

Fig 1.—*A* (Sept 10, 1954) On Aug. 16, 1954, the patient had a modified right upper thoracoplasty. On Sept. 8, 1954, she had right upper lobe lobectomy for tuberculosis. On Sept. 10, 1954, the chest film showed a significant air-fluid collection in the right chest and the tubes were not draining well.

B (Dec. 6, 1954). SK-SD solution was instilled into the chest through the upper and lower tubes. The fluid and air were aspirated three hours later. This was done three times during the first postoperative week. Although the treatment caused considerable pleural reaction, the pleural space was effectively evacuated. Most of the pleural thickening had disappeared by Dec. 6, 1954.



A.

B.

C

Fig. 2—*A* (Nov. 8, 1954) On Oct 4, 1954, a modified right upper thoracoplasty was performed. The patient had a right upper lobe lobectomy and resection of the superior segment of the right lower lobe on Oct. 26, 1954, for tuberculosis A five-rib thoracoplasty was completed at the time of surgery. Postoperatively, he had a persistent apical space despite repeated needle aspirations and the use of indwelling polyethylene catheters A small fistula was present

B (Nov. 12, 1954). *On Nov. 10, 1954, a tube was inserted to the tip of the apical space through the anterolateral chest wall under* direct vision The subscapular space was avoided, and pneumoperitoneum was started. The pleural space was obliterated soon and the fistula closed thereafter

C (Nov. 30, 1954) By Nov 30, 1954, he was convalescing well and there was no recurrence of the space

Attention to details at the time of resection contributes to early evacuation of the pleural space and cessation of air leaks. The lysis of pleural adhesion, especially in the lower half of the chest, permits a free egress of fluid and air through the drainage tubes. It also allows for uniform expansion of this lung remnant. Sometimes, however, it is possible and judicious to leave some of the apical adhesions intact to prevent later space problems in that area.

If segmental surgery is performed, the remaining raw segmental surface should be free of disease. The blunt dissection used during the removal of a segment is difficult if inflammation or fibrosis has crossed the intersegmental plane. Deviations occur that either remove too much or too little parenchyma. Devitalized tissue may be left behind or injuries may be made to the bronchi, veins, or lung tissue of the adjacent and remaining segment. Such technical errors lead to localized pulmonary infarction, expansion difficulties, and troublesome air leaks. It is sometimes wise to remove more lung than planned to prevent the above complications.[2]

After the removal of the specimen, any depleuralized lung surface should be examined carefully for bleeding and large air leaks. These should be properly sutured. Small alveolar leaks usually close over soon. There should be minimal distortion of the lung remnant. If there has not been too much suturing of the raw segmental surface and there are no limiting adhesions, a normal lung remnant should expand evenly and early following surgery. All transected major bronchi should be covered with pleural flaps or adjacent tissue. Complete intrapleural hemostasis will be rewarding since it usually obviates postoperative transfusions, frequent chest taps, and later surgery. Near the end of the resective surgery, all segments of the remaining lung should expand well with positive intratracheal insufflation. Any expansile lag of all or part of the lung may be caused by remaining disease, endobronchial material, or operative kinking of a bronchus. Such a finding is best corrected at the time of surgery.

Proper drainage of the pleural cavity is very important. It is wise to use two No. 40 French tubes. The tip of one should be placed as high in the chest as possible to remove air, and the other should be in the most dependent position to remove fluid. They should be so inserted that limitation of pulmonary expansion does not occur. The external ends of the tubes are attached to water-seal drainage to allow good escape of pleural contents during the closing of the wound and thereafter. To prevent early intrapleural clotting, about 300 c.c. of saline are poured into the pleural space just prior to closure. Antibiotics are added to this fluid to combat early postoperative infection.

Concomitant pneumoperitoneum is a valuable adjunct in resection of all or part of the lower lobe. About 700 c.c. of air is instilled into the abdomen after closure of the chest. More air is added later depending on the condition of the patient and the roentgenographic findings. Preservation of the phrenic nerve prevents troublesome paradoxical motion and difficulty with bronchial secretions. More recently concomitant pneumoperitoneum following upper lobe surgery has been used. The rationale for this is upward displacement of the remaining lung which then expands more readily into the apical space. The first few patients treated in this manner have had no space difficulties, but further studies are necessary for complete evaluation.

ACTIVE EARLY POSTOPERATIVE CARE

Aggressive management shortly after surgery is important to prevent pleural space problems. The tracheobronchial airway should be clear at all times. Usually coughing, deep breathing, and frequent changes of the patient's position suffice. Frequently, however, intratracheal suction by catheter is necessary and occasionally blood, mucus and exudate must be removed by bronchoscopy. A helpful adjunct is the use of the intermittent positive pressure breathing apparatus, which is good for clearing the bronchiolar passages.[3, 4] It may be used in the presence of a bronchopleural fistula provided a pleural drainage tube is in place leading to a water seal bottle or suction.

Prophylactic treatment against pleural infection is important. Appropriate oral and parenteral antibiotics should be started before surgery and maintained for at least a week after operation. This treatment will also combat pulmonary infection provided atelectasis does not develop. Early apposition of the visceral and parietal pleurae is one of the best preventives against empyema. An obliterated pleural cavity rarely develops infection whereas a persistent pleural space is prone to become infected later.

One of the most pressing problems is the maintenance of adequate pleural drainage. Large rubber tubes are the best conduits to remove intrapleural air and fluid. They should lead at least to water seal bottles at steady downward gradients. There are advocates of various degrees of suction ranging from 6 to 40 cm. of water. Usually 6 to 10 cm. of water suction is adequate, but higher degrees may be used later in the recovery period. Low suction does not cause excessive pleural edema or reaction. When all air and fluid have been removed, the air leaks probably close by their apposition to adjacent surfaces and fibrinous occlusion.

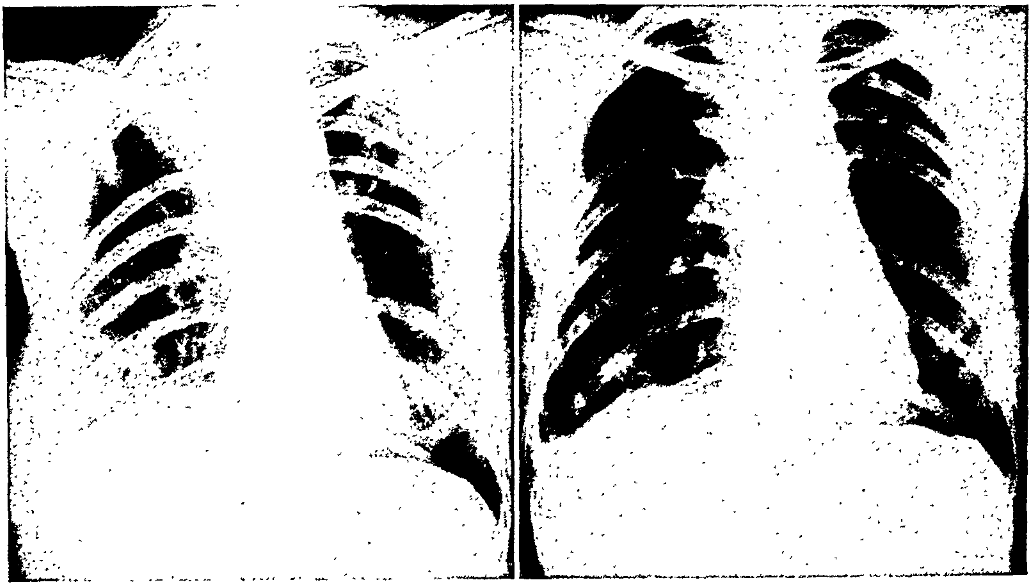
Chest tubes are removed when their fluid columns stop fluctuating and the space is obliterated. This usually occurs from the second to the seventh postoperative day. If a tube is plugged prematurely, it should be milked close to the chest to resume patency. Sometimes, antibiotic solutions or streptokinase streptodornase (SK SD) must be instilled into the tube proximally to dislodge or dissolve clots. If a bronchopleural fistula persists, at least one tube usually stays open and it must be kept open until the desired result is obtained. When expanding lung separates the internal opening of a tube from a persistent pleural space that does not have a negative pressure, the tube should be removed and another inserted to the proper place.

THE PERSISTENT POSTOPERATIVE SPACE AT THE PLEURAL APEX

Although a patient has been treated by the above standards, an apical space may persist. This space may be benign, but it may herald future difficulties. It should be considered dangerous until more is known about it. The alert surgeon should evaluate and treat it as soon as possible.

An obstructed tube, especially to the upper chest, in the presence of an apical space is either plugged with fibrin or the internal opening is closed by the expanding lung remnant. When this occurs, the surgeon should know the pressure within the space. This may be done by tapping with a needle the air pocket posteriorly at the proper level. Any fluid is withdrawn if possible. Air is removed until a high negative pressure develops or until it is obvious that a fistula is present.

When a high negative pressure is maintained, the upper tube can be removed. The lower tube is taken out when it cannot be made to function properly or when the lower chest contains no air or fluid. The apical space is studied by serial roentgenograms. When it progressively diminishes in size without fluid collection, nothing further is done. Films will show a convex upper lung remnant rising toward the dome of the chest. If obliteration is slow or fluid recurs, the space may have to be tapped several more times (Fig. 3, *A* and *B*). Antibiotic solutions should be instilled when infection is feared.



A. *B.*

Fig. 3.—*A* (Sept. 17, 1954). In March, 1954, a segmental resection was performed on the left lung for tuberculosis, and the patient made a good recovery. On Sept. 10, 1954, she had an apicoposterior segmental resection of the right upper lobe for the same disease. It was also necessary to remove a wedge of tissue from the adjacent anterior segment. On the fifth day after surgery an apical space was demonstrated by chest films. For the next five days it was aspirated by needle through the third interspace, posteriorly. No fistula was demonstrated and the space closed slowly but progressively.

B (Nov. 23, 1954). Seventy-four days after surgery her film was as shown.

More complicated treatment is necessary when the initial chest tap shows the presence of a fistula by free flow of air through the needle. The same is true when, at the end of tapping, a negative pressure is reached which becomes neutral or positive the next day, indicating a slow bronchial leak. If both tubes are in place, the surgeon may elect to insert streptokinase-streptodornase through the upper tube (Fig. 1).[1, 4] This should dissolve any intraluminal and adjacent clot so that the upper tube again communicates with the apical space.

Following instillation, both tubes are clamped for three or four hours. Relatively high suction is exerted on them when they are opened. The intrapleural fluid and air is drawn off as fast as possible with the hope that the apical space will obliterate immediately. If this ideal is not obtained, constant suction of about 10 cm. of water is maintained until the tubes cease to function.

Occasionally polyethylene tubing has been used to decompress an apical space that is not intubated effectively. Such treatment is used when the fistula is very small. The tubing is inserted posteriorly through a No. 15 gauge needle into the space and attached to low suction. The space has disappeared with this method in a few cases. Usually this treatment has been unsatisfactory due to plugging of the tube despite frequent cleaning with antibiotic solutions. Apparently the egress of air or fluid from the space often is not fast enough to allow expansion of the lung remnant.

At times, an intubated apical space fails to obliterate despite the above management. The functionless upper tube is then removed and another tube is reinserted immediately. This is preferably done through the third interspace in the anterior axilla. The pectoralis muscle is not injured since its lateral border is retracted forward. A closed procedure using a trocar usually suffices. If lung is adherent to the parietal pleura in this region, the intercostal space is opened for a short distance, the pleural symphysis is parted and the tube is advanced into the pleural apex under direct vision (Fig. 2). The wound is closed tightly in layers about the tube.

Sometimes an apical air space under neutral or positive pressure will lead to retraction of the lower lung as well. The roentgenogram will show two spaces with apparent pleural apposition in the mid lung field. Almost invariably, however, these two spaces communicate. This situation requires both apical re-intubation as described previously and closed intubation of the lower space to ensure fast re-expansion of the lung.

It is probably beneficial to start pneumoperitoneum whenever a space becomes difficult to obliterate if it has not been given already. The adjunct is of value *only when there is a negative pressure in the pleural space*. This often implies the presence of a good functioning intrapleural tube of adequate size connected to low suction, externally.

In some cases, a drainage tube may remain patent for many days without special care. This usually means that there is a persistent space and relatively large bronchial leaks. If the lung remnant is large enough and expansible, pleural obliteration should appear before long. When signs of pleural infection appear, such as purulent drainage or systemic reaction, an adequate thoracoplasty should be contemplated with the drainage tube in situ.

THE PROBLEM OF THE BASAL SPACE

Following resection of all or part of a lower lobe, there is almost always a space in the lower chest during the immediate postoperative period. Because of its dependent position, the problem of fluid evacuation is important. Any fluid should be withdrawn as early as possible before clotting develops. Other aspects of blood in the lower chest will be discussed later.

The use of concomitant pneumoperitoneum at the time of lower lobe surgery has been most beneficial, and it is used whenever possible. The elevation of the diaphragm diminishes the size of a basal air space, and less expansion of the lung remnant is required to close it. It is re-emphasized that the space is best obliterated by proper intubation with suction.

Rarely, the upper lung remnant is too small or it has unrecognized disease so that it will not expand into a basal space. If good drainage and pneumoperitoneum are of no avail, an upper thoracoplasty or plombage may be performed. The lung is shifted downward by these operations and less expansion is needed to fill the basal space.

A pleural space problem in the lower chest worthy of special mention is that which appears next to the mediastinum. Such spaces usually contain both air and fluid, and the fluid level seen by roentgenography may not extend to the chest wall anywhere. Paramediastinal air and fluid collections usually develop originally by the dissection of air from a bronchial leak or an undrained air pocket elsewhere in the same pleural cavity. If such a space is inadequately treated, it will soon loculate. Tapping the area may injure adjacent lung and the placement of a drainage tube would have to be in an awkward location.

It is recommended, therefore, that an open thoracotomy be done for a paramediastinal collection that does not respond to existing drainage (Fig. 4). This surgery is done preferably before the lung is firmly adherent to the diaphragm or before ten postoperative days have elapsed. A small incision is made in the chest posterolaterally at the level of the diaphragm. If possible, the pleura is entered through an intercostal space, but it may be necessary to remove a short segment of rib. The lower surface of the lung is separated from the diaphragm until the paramediastinal space is entered. A large soft tube is inserted then with the aid of a trocar through the chest wall near by so that its tip rests at the base of the space near the mediastinum. All fluid is removed under direct vision. The original thoracotomy wound is closed and the tube is attached to low suction.

To prevent this complication from reappearing, the surgeon should decompress any inadequately treated air space or bronchial leak elsewhere in the pleural cavity. This may require the insertion of another drainage tube to the proper site and again low suction should be used (Fig. 4, *B* and *C*).

HEMOTHORAX IN THE EARLY POSTOPERATIVE PERIOD

A certain amount of bloody fluid always appears in the pleural cavity following pulmonary resection, and most of it collects during the first twenty-four hours. This creates an abnormal pleural space and the contents should be evacuated before clotting occurs. The installation of 300 c.c. of saline prior to closure delays early clot formation. Dependent constant drainage from the time of surgery usually gives good results.

When there is persistent intrapleural fluid and little or no drainage from the chest tube, thoracentesis should be done as often as necessary until only a small amount remains. Early needling, especially during the third or fourth

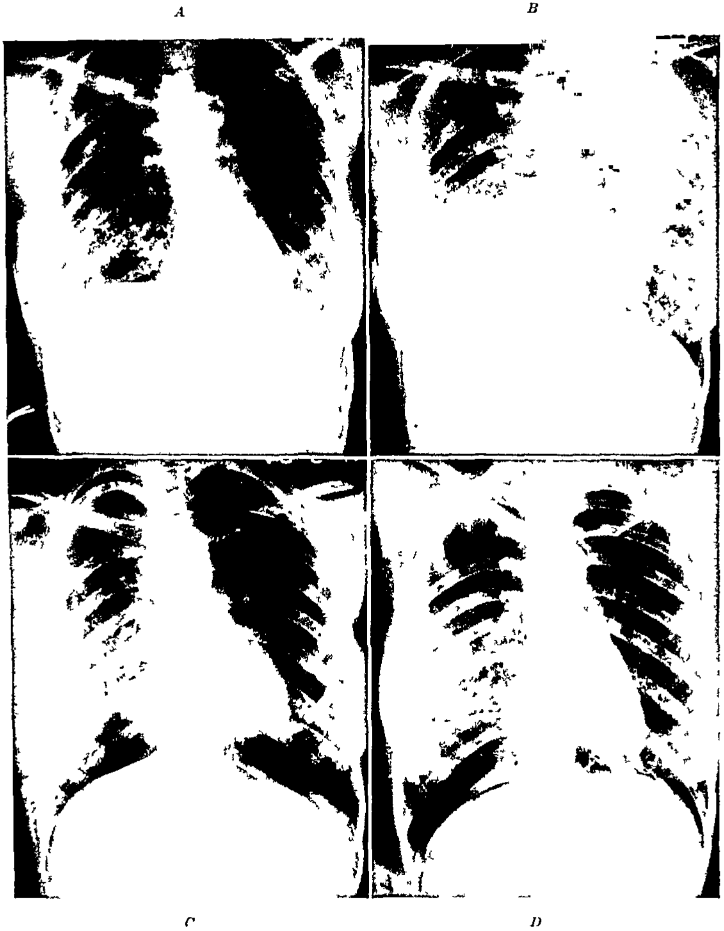


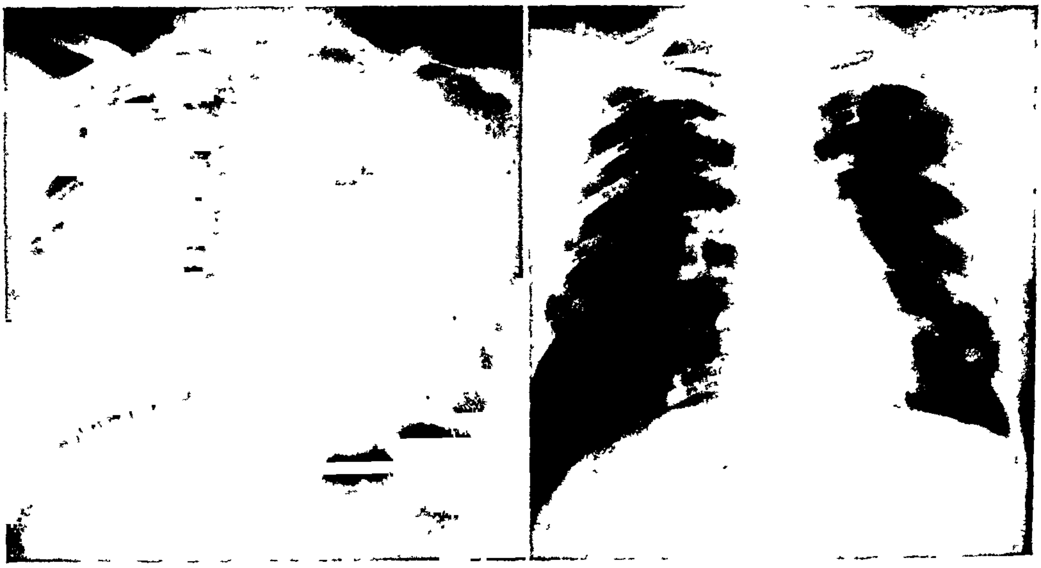
Fig 4—*A* (Nov 5, 1954) On Oct 31 1954 the patient had an apicoposterior segmental resection of the right upper lobe for tuberculosis By the fourth day after surgery, both upper and lower tubes were plugged and there was an apical space The lower tube was removed On Nov 5, 1954, besides the apical space there was a paramediastinal air-fluid collection above the diaphragm SK-SD was instilled through the upper tube Some of the fluid and air was aspirated through the tube by high suction, but the fluid collection in the lower chest could not be removed by thoracentesis This complication might have been avoided if the lower tube had not been removed

B (Nov 7 1954) On Nov 7 1954 there was a large supradiaphragmatic and paramediastinal collection The apical air space was still present On the same day a tube was inserted through the bed of the seventh rib to the paramediastinal space, and the apical space was intubated again Pneumoperitoneum was started

C (Nov 9, 1954) Roentgenograms taken two days later showed marked improvement

D (Dec 10, 1954) Forty days after surgery, the pleural reaction had almost disappeared postoperative day, gives the best results. Once clotting occurs, several installations of SK-SD solution through one or more chest tubes have been beneficial (Fig. 5). There must be at least one tube in a dependent intrapleural position. Once fibroplasia has started, disappointing results are seen with this agent. It is best, therefore, to use it during the first postoperative week. The ideal result must show the entire superior surface of the diaphragm and completely expanded lung by roentgenography.

Occasionally, none of these procedures gives a good result. To prevent complications arising from a significant hemothorax, it is sometimes necessary to re-enter the chest through a short incision, evacuate all clots and fluid, and reinstitute closed intrapleural drainage. It is frequently easier to obtain a good result by this procedure than by decortication later. If evacuation is done before ten postoperative days have passed, a restricting visceral peel has not developed.



A. *B.*

Fig. 5.—*A* (March 29. 1954). On March 26, 1954, the patient had an apicoposterior segmental resection of the left upper lobe for tuberculosis. The pleural space was obliterated by adhesions and an extensive dissection was necessary to free the lung. Postoperatively there was considerable bleeding into the left chest. The chest film was as shown three days after operation. With both drainage tubes in place, SK-SD, tracheal suction, and intermittent positive pressure breathing were used repeatedly with gratifying results.

B (Aug. 11, 1954). Four and one-half months later the above roentgenogram was taken.

There is an ominous type of hemothorax that usually occurs during the first twenty-four hours after surgery. Persistent bleeding into the pleural space from a small vessel or vessels may lead to early clotting. The drainage of blood from the chest may be alarming but incomplete, and the problem of blood replacement becomes acute. The blood pressure becomes unstable despite repeated transfusions. The lung remnant is compressed and the mediastinum may shift to the opposite side. Changing the patient's position causes sudden hypotension. Before this clinical picture develops completely, re-exploration is mandatory with the evacuation of pleural contents and the ligation of bleeders.

THE APPEARANCE OF LATE PLEURAL SPACES AFTER RESECTION

Pneumothoraces may appear ten or more days after surgery. Some are seen as better roentgenograms or different projections are obtained. Although early chest films may have showed complete expansion, a thin air space was probably present. At any rate, the sudden appearance of a large space indicates a bronchopleural fistula, and prompt closed intubation should be done. Low suction to the drainage tube usually leads to pleural obliteration and closure of the fistula (Fig 6).

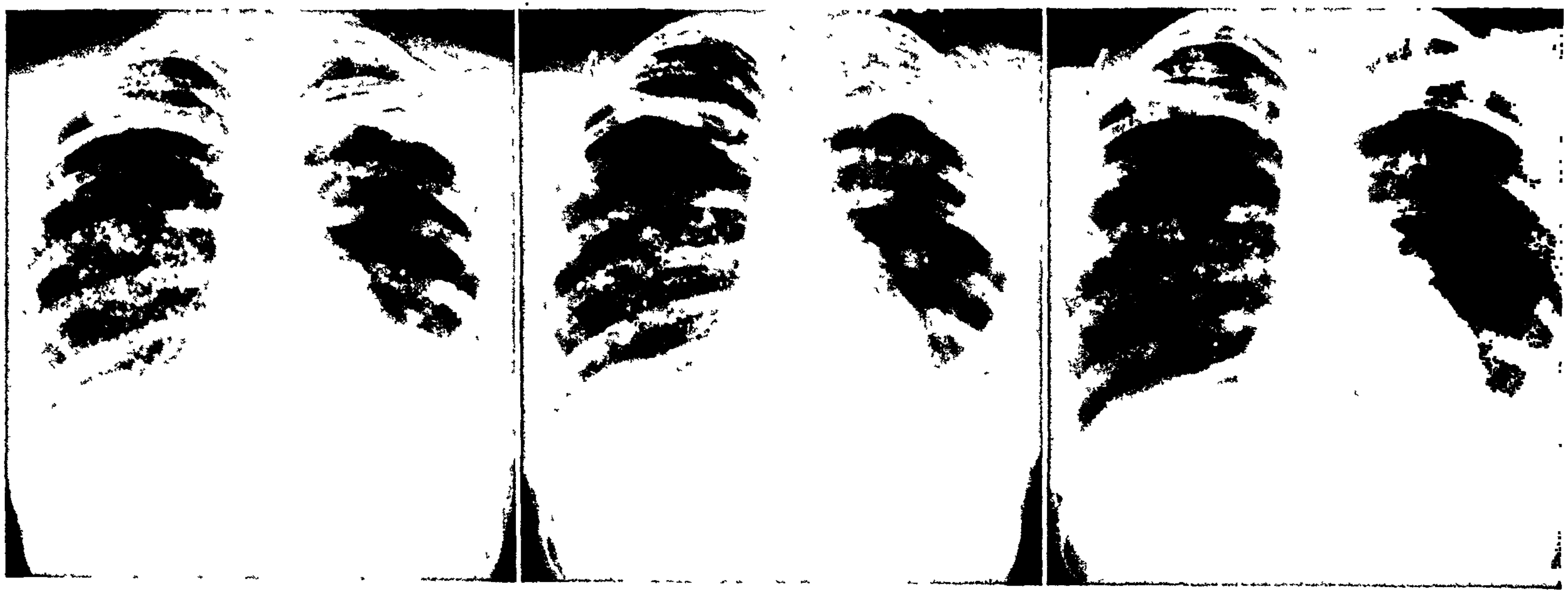
MANAGEMENT OF PERSISTENT AIR SPACES DESPITE ABOVE TREATMENT

A large air space that persists without improvement over two weeks after surgery often implies inadequate treatment earlier. If antibiotics have been used, empyema usually has not developed even at this interval. At least systemic signs and symptoms of pleural infection are rare. The surgeon knows, however, that empyema is a constant threat, and he must get rid of this space as soon as possible. The procedures that must be used are more extensive than those previously mentioned. Generally, the longer necessary surgery is delayed, the greater will be the ultimate loss of pulmonary function.

When an air space persists without leakage from the lung remnant, it usually is situated in the upper half of the chest. With needle aspirations, high persistent negative pressures can be obtained. The upper portion of the lung, however, may not expand appreciably because there is a limiting visceral peel or there is disease within the lung remnant. An extensive and thick peel over the visceral pleura must be decorticated if the underlying lung is to increase in volume, and it may be necessary to excise the parietal surface of the space as well. If it is inadvisable to open the chest, a modified upper thoracoplasty may be beneficial. Frequently this is a safer operation. The number of ribs to be removed will depend on the size of the space to be closed. Usually a first stage procedure suffices, and in the absence of a fistula, the space may be aspirated by needle through the parietal wall at the time of surgery.

When an apical space of any size persists due to a bronchopleural fistula, it must be intubated with the application of low suction (Fig 7). The tube is best inserted in the third interspace as far to the front as possible but lateral to the pectoral muscle. After such drainage has been established, a modified thoracoplasty should be done taking care to avoid the tube tract. All efforts should be made to avoid contamination of the subscapular space. Again a first stage operation usually is adequate, but on occasions the posterior segments of more ribs must be removed later. The establishment of drainage and thoracoplasty can be done concomitantly, but in the order mentioned. This surgery should not be put off too long, for progressive thickening of the parietal pleura may eventually prevent good collapse.

Persistent air spaces with bronchopleural fistulas in the lower chest that have received some treatment are usually not large. If they are to be eliminated, intubation with suction is necessary. The diaphragm should be elevated as high as possible by pneumoperitoneum. Sometimes a very small space



A. *B.* *C.*

FIG. 6.—*A* (Nov. 8, 1954). On May 11, 1954, the patient had an apicoposterior segmental resection of the right upper lobe for tuberculosis and she made a good recovery. On Oct. 6, 1954, the same operation was done on the left upper lobe. There was a space problem during the early postoperative course requiring needle aspiration and secondary intubation. The left pleural cavity seemed to be obliterated until four weeks after surgery when an apical space appeared. On Nov. 8, 1954, there was an air-fluid level in the left upper chest which could not be remedied by tapping although a fistula was not apparent by needle aspiration.

B (Nov. 16, 1954). A catheter was inserted into the space under direct vision through the anterior axilla with good expansion of the lung remnant by Nov. 16, 1954.

C (Nov. 27, 1954). By Nov. 27, 1954, the remaining left lung had expanded well into the apex.

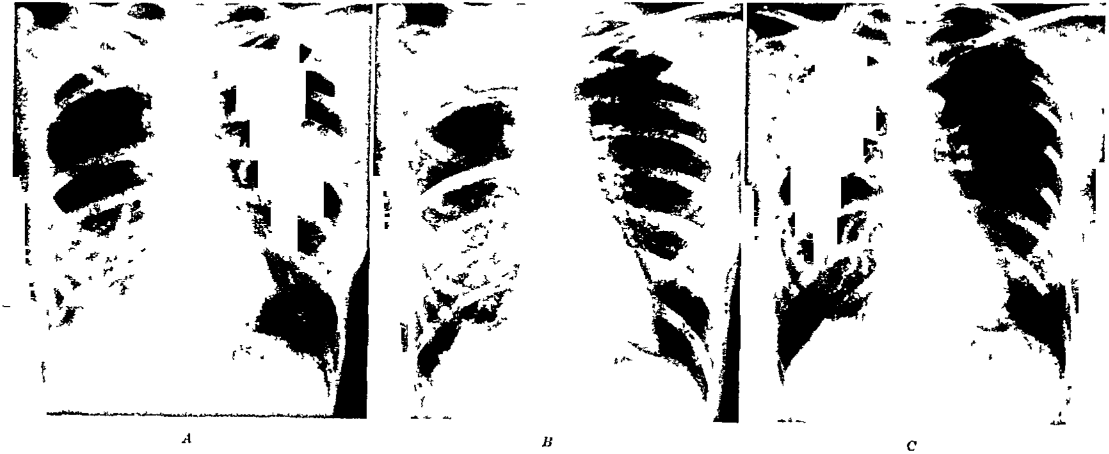


Fig 7 —*A* (July 15 1954) On June 30 1954 the patient had an apicoposterior segmental resection for tuberculosis At first her roentgenograms looked well but she later developed an apical space with evidence of a small fistula Repeated needle aspirations were of no avail

B (Aug 17 1954) On July 27 1954 a modified right upper thoracoplasty was performed following intubation of the space The space and fistula were closed without recurrence

C (Dec 10 1954) Twelve weeks after resection her chest film is as shown still remains, usually because of leakage about the tube. It may then be wise to divide the tube at skin level and resort to open drainage. The residual pleural cavity is allowed to contract with repeated changes to smaller drainage tubes. Ultimate obliteration of space and fistula may occur. When this does not happen, the resection of more lung to include the fistula may be necessary after several months have elapsed.

SUMMARY

A persistent pleural space following partial resection of the lung may lead to empyema and bronchopleural fistula sometimes long after resective surgery. Prompt care must be given to obliterate the space soon after resection. Infection is usually absent at this time. The visceral pleura is pliable, and transected lung surfaces expand more readily. When this golden period of management is allowed to slip by and a pleural space persists, various processes hinder a satisfactory outcome. The depleuralized surface of lung and the visceral pleura become leathery and thick, impeding inflation of the lung remnant. Fistulous openings become more rigid, and they may not close when brought against another surface. Persistent air leaks maintain neutral or positive intrapleural pressures and hinder pulmonary expansion further. The hazard of infection increases daily.

Presented here are methods of management of the postoperative pleural space with illustrative case reports. Although therapeutic generalities are discussed, each patient must be treated according to his own local and general problems.

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FOREIGN BODY BRONCHIECTASIS

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DURING a recent review of the experience of the Mayo Clinic in bronchiectasis, it appeared that bronchial disease secondary to foreign bodies manifests certain different clinical and prognostic characteristics. It was with this observation in mind that separate consideration was given to foreign body bronchiectasis.

MATERIALS AND METHODS

This study consists of 14 cases in which bronchiectasis apparently was caused by an obstruction of the bronchus and was secondary to a foreign body. The 14 patients were treated surgically from 1940 through 1953. Bronchoscopic, bronchographic, and pathologic studies were made in each case. Follow-up studies were possible in all 14 cases. Pulmonary resection was carried out in all instances with the aid of general endotracheal anesthesia, and the procedure consisted of a posterolateral thoracotomy incision and individual ligation of hilar structures. Pathologic examination confirmed the clinical impression of bronchiectasis in all cases, and in each case revealed the foreign body.

CLINICAL FEATURES

The distribution of these 14 patients according to age is given in Table I. Most of them were in the younger age groups. There were 8 males and 6 females in the series. All the patients manifested chronic productive cough. The occurrence of hemoptysis (8 patients or 57 per cent) was in accordance with the findings of others reporting cases of foreign body bronchiectasis.[1,2] The interval after aspiration of the foreign body ranged between six months and

TABLE I. INCIDENCE ACCORDING TO AGE. 14 PATIENTS WHO HAD FOREIGN BODY BRONCHIECTASIS

AGE (YEARS)	PATIENTS (NO.)
0-9	1
10-19	5
20-29	5
30-39	1
40-49	2

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twenty years. The exact distribution of this interval is given in Table II. The location of the foreign body was equally divided between the two sides, there being seven foreign bodies on each side.

TABLE II. INTERVAL BETWEEN ASPIRATION OF FOREIGN BODY AND OPERATION: 14 PATIENTS

INTERVAL (YEARS)	PATIENTS (NO.)
0-5	3
6-10	1
11-15	6
16-20	3
Not known	1

The lower lobe of the lung was involved in each case. No isolated instance of involvement of the upper lobe was found; however, in 5 cases involvement of the upper lobe accompanied involvement of other lobes of the lung, thus requiring pneumonectomy. In the remaining cases, unilateral unilobar resection was done in 8 of them, and unilateral multilobar resection (right middle and lower lobes) was done in 1.

RESULTS

There were no surgical deaths. An excellent result was obtained in every case. We regard an "excellent result" as one in which the patient has little if any residual cough and expectoration, and has obtained full functional rehabilitation.

Results of our study are in striking contrast with the clinical results obtained for 221 patients with primary bronchiectasis treated surgically at the Mayo Clinic during the same period; in that series an excellent result was obtained for 75 per cent of the patients.[2] Of the 18 patients in the group with primary bronchiectasis who underwent pneumonectomy, 14 (78 per cent) obtained a good result. This higher incidence of excellent results obtained for those who underwent pneumonectomy for primary bronchiectasis may partially explain why surgery produced such an excellent result for our patients with foreign-body bronchiectasis, since 5 of the 14 in this group underwent pneumonectomy. Furthermore, at operation it was noted that 6 of the patients with foreign-body bronchiectasis had marked contraction of the affected lobes. It is probable that the patients in this group already had made a physiologic adjustment to the loss of pulmonary tissue, and this may be another explanation of the good results obtained from surgery.

The postoperative complications were five instances of empyema and of hemothorax requiring two thoracenteses. It is interesting and evident that a good result may be obtained despite the occurrence of postoperative empyema.

There was no instance of postoperative atelectasis in these 14 cases, nor was there any retention of secretions sufficient to require bronchoscopy. Here, again, results were in striking contrast to those in the series of primary bronchiectasis[2] in which 25.8 per cent of the patients required bronchoscopy for retention of secretions.

CONCLUSIONS

The prognosis after pulmonary resection for foreign body bronchiectasis is good An excellent clinical result was obtained for all patients with foreign-body bronchiectasis treated surgically, as opposed to excellent results obtained for only 75 per cent of patients who had primary bronchiectasis Postoperative atelectasis, occurring in 25 8 per cent of patients who had primary bronchiectasis, did not appear in 14 who had foreign-body bronchiectasis By contrast, the incidence of postoperative empyema among those who had foreign body bronchiectasis was much more (30 per cent) than it was among those who had primary bronchiectasis (6 per cent) We attribute this difference to the increase in obstructive pneumonitis in the presence of foreign body bronchiectasis

On the basis of the foregoing data, it is apparent that the prognosis after surgical resection for foreign body bronchiectasis is much better than it is after surgical resection for the primary form of the disease This would seem to indicate that there are basic pathogenetic and physiologic, if not pathologic, differences inherent in foreign body bronchiectasis that distinguish it from primary bronchiectasis Early bronchoscopic aspiration of foreign bodies is important in the prophylaxis of foreign body bronchiectasis,[3, 4] whereas surgical resection would appear to be the treatment of choice when the condition has already developed

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EXPERIMENTAL REPLACEMENT OF THE SUPERIOR VENA CAVA WITH HOMOLOGOUS ARTERIES, AND REPORT OF A CASE WITH MALIGNANT OBSTRUCTION REPLACED WITH A HETEROLOGOUS ARTERY

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INTRODUCTION

ACUTE obstruction of the superior vena cava, though infrequently encountered, is of clinical importance because of the discomfort and incapacitating effects on the patient. In a review of the subject,[1] malignancy was reported to have caused 44 per cent of the obstructions; aortic aneurysm, 32 per cent; and undetermined origin, believed to be a form of mediastinitis, 12 per cent. In another series,[2] signs and symptoms of superior caval obstruction were present on admission in 2.2 per cent of patients with proved carcinoma of the lung.

There have been occasional reports of surgical attempts to relieve the obstruction.[3-9] A patient with superior caval obstruction caused by a malignancy may be treated by external radiation. Relief of the symptoms is often obtained. If surgical exploration is indicated for possible palliative resection of the lung, and if technical factors are favorable for resection and grafting, removal of the involved vessel and replacement with a graft now appears to be the procedure of choice. Immediate relief of symptoms can be expected and there is the likelihood of making the patient more comfortable for a significantly larger percentage of his life expectancy.

Good results have been reported with autogenous femoral vein grafts used for the replacement of the superior cava.[6-8] Both viable and nonviable homologous arteries have also been used for this purpose with success.[7-9] Homografts seem to be more likely to succeed when preserved by the freeze-drying process than when in the viable state, judging from small differences observed between the results of series of freeze-dried and comparable fresh homografts in the abdominal aortas of dogs.[10, 11]

Autogenous vein is probably the material of choice for caval replacement. The additional incision required is a disadvantage. Extensive studies have not been done to determine the relative incidence of thromboses in autogenous vein grafts versus homologous arteries implanted in veins. However, a greater

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tendency toward thrombosis has been demonstrated in freeze-dried homologous arteries than in free autogenous carotid grafts in the dog[1] This is a region where the tendency to thrombosis in homografts is high because of the small size of the vessel[1-] Minimal foreign tissue reaction may cause an occlusive thrombus in a small vessel or one in which flow is slow

Freeze-dried arterial heterografts offer another possible solution to the problem They have been found to be more successful than fresh heterografts when used in the abdominal aortas of dogs A freeze-dried vessel contains less soluble protein material than a fresh unextracted one[13, 14] Successful results using freeze-dried bovine and porcine heterografts in a short series of human cases were reported by Hufnagel[15]

While some woven plastic prostheses are satisfactory in large and medium sized arteries, experimental work in animals has made them appear extremely unpromising for the superior vena cava[1] Though shunts are useful, and probably essential in some locations to maintain circulation during the resection and grafting of certain segments of the aorta,[1, 18] there appears to be no indication for the use of a shunt around a vein which is largely or completely obstructed at the time of exploration

Animal experiments have been undertaken to study the use of freeze-dried homologous arterial grafts in the superior vena cava

METHOD

The descending thoracic aorta was removed from 45- to 60 pound dogs, using sterile autopsy technique This tissue was freeze-dried according to the method of Pate[10]

Right thoracotomy using aseptic technique was performed on dogs under intravenous Nembutal anesthesia and 100 per cent endotracheal oxygen under intermittent positive pressure The superior vena cava was occluded at its origin with a rubber-shod Rankin hemostat and the pressure on the vessel controlled by a rubber band around the handle A V shaped portion of the right superior aspect of the right auricle, near the base of the auricular appendage, was occluded with Potts' clamps After the cava was resected, the graft was sutured first to the upper end of the cava using two stay sutures, connected by continuous everting mattress sutures of No 5 0 silk The graft was then sutured to the auricle in like manner, with careful eversion of the auricular edges No anticoagulants or antibiotics were used postoperatively

RESULTS

There were no early postoperative deaths Of the 6 dogs operated on, 1 died after four weeks At autopsy, the pleural cavity was found to be largely filled with fluid, and the graft was completely thrombosed The other 5 dogs were sacrificed after 1, 1½, 4, 5, and 5 months In all cases, each graft was the same size as when implanted, with no evidence of excessive fibrosis around the outside or mural thrombus on the inside The suture line between the superior vena cava and the graft was well endothelized and without stricture However, the anastomosis between the graft and the right auricle was found to contain a thin diaphragmlike stricture in all cases. The opening was only one-third to one-fourth the original diameter of the graft. The stricture appeared to have formed in the auricle immediately adjacent to the anastomosis and may have been the result of an organized thrombus.

Cephalad venous pressure was elevated in all cases after approximately two weeks, and significant pleural effusion was found at autopsy in 2 of the dogs. Microscopic examination of the grafts showed good endothelization, loss of the muscular elements with persistence of the elastic fibers, and invasion by host fibroblasts. This picture was similar to that obtained with freeze-dried homografts in the abdominal aorta.

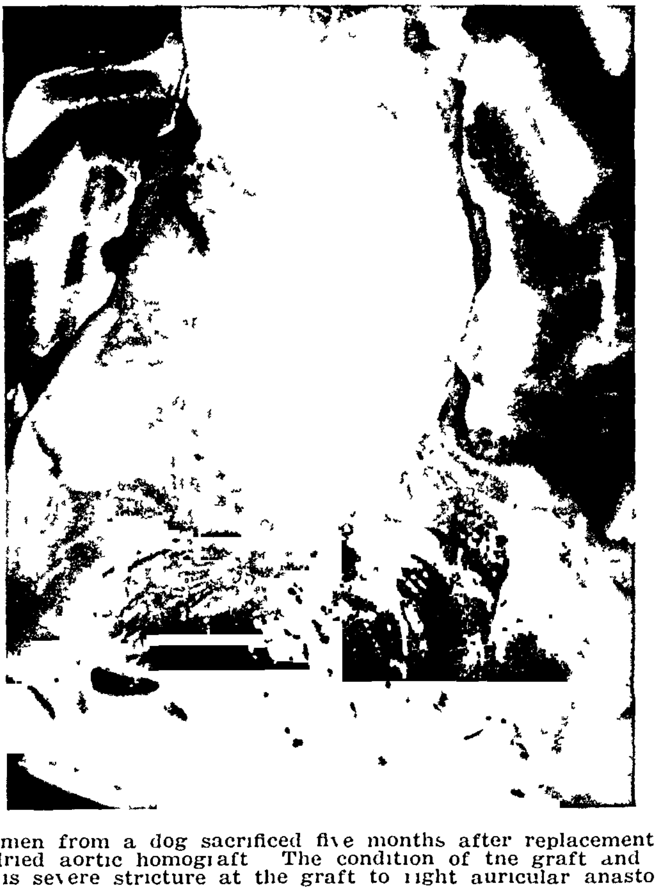

Fig. 1.—Specimen from a dog sacrificed five months after replacement of superior vena cava by a freeze-dried aortic homograft. The condition of the graft and upper suture line is good, but there is severe stricture at the graft to right auricular anastomosis.

DISCUSSION

These results would seem to suggest that a graft to cava anastomosis is likely to succeed, whereas one between the graft and the auricle is likely to stricture. These strictures at the auricular anastomosis are similar to those noted in some cases by Gerbode[19] and others[7] after transplantation of vessels to the right auricle.

The first successful treatment of superior vena caval occlusion by arterial graft was reported by Holman and Steinberg in 1954.[9] In their case, occlusion was caused by nonspecific mediastinitis and the occlusive process involved the entire vena cava save a small segment at either extremity, thereby enabling them to use a freeze-dried arterial homograft in graft to cava anastomosis rather than graft to auricle. Their patient has done well since operation and certainly (to use the words of Holman and Steinberg[9]) "the desperate plight of patients with such an obstruction warrants continued trial of this procedure."

CASE REPORT

A case is reported illustrating the possibility of obtaining a successful result with a freeze-dried porcine aortic graft, when surgical replacement is desired and alternative procedures are not feasible for one reason or another.

This is the case of a 69-year-old white man, who was admitted to Doctors Hospital on March 26, 1955, complaining of swelling of the face, neck, arms, and chest, for one week. He stated that while on his yacht about a week prior to his admission, March 17, 1955, he awoke one morning to find his face markedly swollen and blue, as well as the arms and upper chest. The veins and venous pattern on the chest were very prominent and the veins in the neck stood out like large cords.

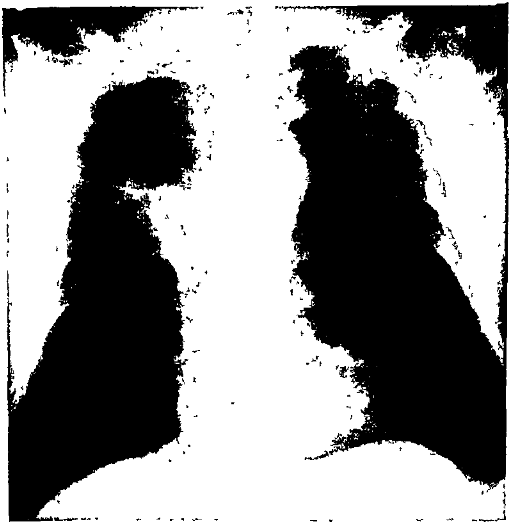

Fig. 2.—Preoperative chest x-rays showing lesion in the right hilum and the cyst noted previously in the right upper lobe.

For several years he had complained of severe cough. However, this had become more severe within the last six months, and at times he was seized with severe paroxysms lasting several minutes. He had never coughed up blood and was not hoarse, and he had no difficulty in swallowing. In the past, he had been seen repeatedly by his internist because of a chronic peptic ulcer, and chest x-rays made in 1950 and 1951, both showed a large cyst in the right upper lobe. He was acquainted with this fact but had had no treatment for this condition. His family history was nonrevealing.

At the time of his admission, he was an obese white man whose face and whole upper body were cyanotic with many prominent veins and venules present over the chest and back. The venous pressure was markedly elevated in the upper extremities, as evidenced by the veins in the hand which remained completely engorged even when the hands were elevated high above the head. He was most uncomfortable and felt the engorgement of his face and head very keenly, and was also somewhat dyspneic. The heart was slightly enlarged to the left and there was a systolic apical murmur. The percussion note over the right upper lobe was somewhat more resonant than normal; however, no dullness could be elicited. Moist râles were heard in the right lower chest. No breath sounds were heard in the right apex. There was no palpable adenopathy and examination of neither the abdomen nor the remainder of the body was remarkable. The blood pressure was 164/80, pulse, 82, and the temperature was 98.6° F.

An x-ray examination of the chest, including fluoroscopy and laminographic studies, showed the presence of a large soft tissue mass apparently arising in the mediastinum near the tracheal bifurcation and projecting into the upper half of the right lower lobe, partial collapse of the right lung, and moderately severe tortuosity of the aorta with calcification of the arch. The mass projecting into the right lower lobe was nonpulsatile on fluoroscopy. Barium swallow revealed localized posterior displacement of the esophagus on the posterior aspect of the heart consistent with enlargement of the left atrium. Laminographic studies contributed little other information except to indicate that there was widening of the right main stem bronchus probably indicating pressure by the mass on the bronchus from behind. There was no evidence suggesting dilatation of the aorta.

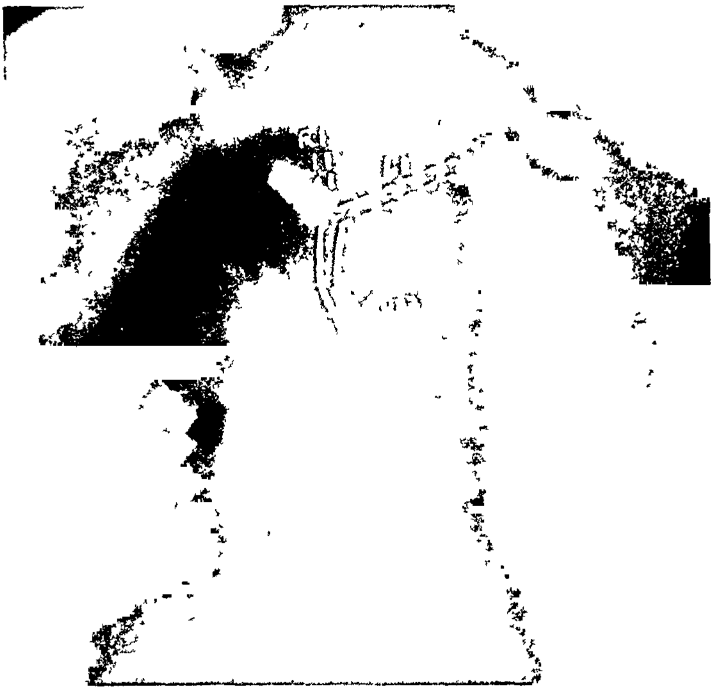

Fig. 3.—Postoperative film, with outline of graft indicated.

On March 19, 1955, he was bronchoscoped, and it was noted that the trachea was fixed in the midline and the carina was greatly widened. Both main-stem bronchi were decreased in caliber; however, no gross tumor was visualized. Bronchial washings were made and sent to the laboratory for study. These washings failed to reveal contributory findings.

A diagnosis was made of bronchogenic carcinoma, probably not resectable, plus a complete block of the superior vena cava. On March 23, 1955, a right exploratory thoracotomy was done and a large mass was found in the right hilum, completely occluding the superior vena cava and surrounding both bronchi Frozen sections made from the lesion revealed anaplastic carcinoma, probably of bronchogenic origin

The superior vena cava was involved in this mass almost throughout the extent of the vessel *At its origin, from the innominate veins, it was possible to dissect a segment* of uninvolved cava for about 1 5 cm This gave an adequate cuff for anastomosis at this site The growth involved the site of entrance of the azygos vein into the superior vena cava as well as the pericardium at the point of entrance of the superior vena cava into the pericardial sac Fortunately, however, when the pericardium was opened it was possible to dissect a cuff of vena cava approximately 1 cm in length, which proved adequate for anastomosis of the proximal end of the graft Noncrushing clamps were used to occlude the cava at both extremities and the vessel was divided first at its upper extremity A suitable length of reconstituted freeze dried porcine aorta was selected for use as the graft to bridge the defect in the cava, and the anastomosis was accomplished by continuous everting mattress suture of No 5 0 silk The intrapericardial portion of the cava was divided and the *remaining end of the graft was sutured to the auricular stump of the superior vena cava* by the same technique No clamp was applied to the graft at any time The remaining cut ends of the involved cava did not bleed and the involvement was so extensive that no attempt at resection of the mass or vena cava was attempted The uppermost clamp was removed first and as the graft filled with blood, the air it contained was displaced through the suture lines, however, very little blood leaked through and this soon ceased completely

When the lower occluding clamp was removed, adequate blood flow occurred through the graft and the anesthetist reported prompt improvement in the color of the patient's face and neck The following day, the patient's edema and abnormal venous pattern over the upper body had disappeared He left the hospital on the seventh postoperative day and has been undergoing roentgen therapy since his discharge *He has returned to work, and has had no* recurrence of swelling of the face and upper body seven months after operation

SUMMARY AND CONCLUSIONS

1 Superior vena cava obstruction caused by malignant invasion is infrequent but constitutes a definite clinical problem

2 Radiation therapy is often satisfactory and is advised if palliative resection is not desired

3 Freeze dried arterial homografts were used experimentally for replacement of the superior vena cava in 6 dogs The cava to graft anastomosis and the graft itself were successful, but the graft to right auricular anastomosis resulted in stricture

4 A case is reported in which a freeze dried porcine aortic graft was sutured to stumps of the superior vena cava at each end with a satisfactory result

5 Resection and replacement by a graft, of a cava obstructed by malignant disease is advised if exploration is indicated for possible resection of the primary tumor, and the graft can be sutured to vein at each end

6 Indications for grafting are less definite when exploration is not otherwise indicated, or when the graft can be sutured only to the right auricle proximally

7 Materials which can be used with success for replacement of the resected cava, in descending order of preference, are autogenous femoral vein, freeze dried homologous artery, and freeze dried porcine aorta

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POSTOPERATIVE COMPLICATIONS RELATED TO PREOPERATIVE ANTIMICROBIAL THERAPY AND OTHER FACTORS

A Review of 600 Consecutive Pulmonary Resections for Tuberculosis*

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Over the years since 1944, when the first pulmonary resection for tuberculosis was done at the Nova Scotia Sanatorium, it has been intriguing to speculate upon the reason why one patient would encounter a postoperative complication whereas some other, apparently similar person, would not Recently there have been a few scientific papers presented suggesting that prolonged preoperative antimicrobial therapy has led to the development of drug resistance in many of the patients and so is responsible for ensuing complications

In an attempt to evaluate the situation as it exists at the Nova Scotia Sanatorium, it was determined to analyze the first 600 pulmonary resections for tuberculosis done there between Nov 14 1944 and March 25, 1955

As postoperative complications of resection the following were accepted (a) early bronchopleural fistula (within two weeks of operation), (b) late bronchopleural fistula (c) empyema, (d) spread of disease (contralateral and homolateral), (e) persisting pleural space, (f) massive wound disruption (2 cases only without above mentioned additional complications), and (g) early death (2 cases only cause unknown)

Table I indicates the age groups and sex of all patients and also of those who developed any of the complications just mentioned It had previously been noted, but not confirmed statistically, that complications were much more

Table I Age Groups and Sex of All Patients Undergoing Resection With Age, Sex, and Percentage of Each Sex Group Developing Postoperative Complications

AGE GROUPS*	ALL CASES			CASES WITH POSTOPERATIVE COMPLICATIONS					
	MALE	FEMALE	BOTH SEXES	MALE		FEMALE		BOTH SEXES	
	NUMBER	NUMBER	NUMBER	NO	%	NO	%	NO	%
0 9	3	1	4	0	–	1	100	1	25
10 19	19	47	66	6	31	8	17	14	21
20 29	82	159	241	23	28	30	19	53	22
30 39	59	99	158	18	30	22	22	40	25
40 49	64	47	111	27	42	7	15	34	31
50 59	11	8	19	7	64	1	12	8	42
60 69	1	0	1	0	–	0	–	0	–
All ages	239	361	600	81	34	69	19	150	25

*Youngest 7 years 10 months oldest 64 years 10 months

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*From Nova Scotia Sanatorium Kentville Nova Scotia likely to occur in men than women; in this series they occurred in 34 per cent of the men as compared to 19 per cent of the women, in spite of the fact that almost 50 per cent more women than men underwent pulmonary resection. Although the numbers are rather small, the tables would also seem to suggest that men in the older age groups, 40 to 49 years and more especially 50 to 59 years, were more prone to complications. When the sexes were combined, the complication rate was roughly 25 per cent, increasing over the age of 40 years, the increase being due to the male patients alone.

The next problem was to attempt to determine if the extent or magnitude of the operation influenced the preponderance of complications postoperatively.

Table II would suggest that the greater the amount of lung tissue removed, the greater the chances of developing postoperative complications, there being 43 per cent complications among the pneumonectomy group, but only 3 per cent among those undergoing wedge resections. The lobectomies represented the average for all operations, 25 per cent. In some of the groups the numbers of operations done were too small to be significant.

TABLE II. THE TYPE OF OPERATION PERFORMED RELATED TO POSTOPERATIVE COMPLICATIONS

TYPE OF OPERATION	NUMBER DONE	ALL COMPLICATIONS		EARLY FISTULA	LATE FISTULA	EMPYEMA	SPREAD OF DISEASE	STUBBORN SPACES
		NO.	%	NUMBER	NUMBER	NUMBER	NUMBER	NUMBER
Pneumonectomy	92	40	43	24	2	23	17	24
Lobectomy	280	69	25	41	0	38	24	44
Bilobectomy	20	6	30	1	1	3	2	3
Lobe + segment	39	16	41	14	0	12	3	11
Lobe + wedge	10	1	10	0	0	0	0	1
Lobe + segment + wedge	2	1	50	1	0	1	0	1
Segmental resection	48	11	23	6	0	4	2	8
Segment + wedge	3	1	33	1	0	1	0	0
Multiple wedge	9	2	22	0	0	0	2	0
Wedge only	97	3	3	1	0	2	1	1
All cases	600	150	25	89	3	84	51	93

Complications, of course, were frequently multiple, bronchopleural fistula often being followed by or preceded by empyema.

The presence of bronchial tuberculosis would be expected to influence the development of postoperative complications, especially bronchopleural fistula. As a matter of policy, no patient was passed for surgery before bronchoscopy had been carried out. If a definite active endobronchial tuberculosis was discovered, antimicrobial therapy was continued until subsequent bronchoscopic examination revealed that the process had healed. Unfortunately, a mucosal lesion may heal very much more quickly than the submucosal disease, but there is no way to determine the state of the submucosal tissues. During the later years of resectional surgery here, the surgeon made special notes and the pathologist reported especially upon the state of the resected bronchial tree where a major bronchus was severed.

Table III presents information about the relationship of tuberculous tracheobronchitis and postoperative complications. Thirty-two patients who had evidence of gross tuberculous tracheobronchitis at the time of operation, and 33 persons with microscopic evidence of active endobronchial disease, and 44 who had shown a prior lesion bronchoscopically, did not develop any post operative complications Evidently, therefore, this preoperative condition need not necessarily lead to postoperative disaster, especially if re expansion of re maining lung tissue may be brought about promptly to cover the stump and if surgery is done under adequate antimicrobial cover It is of interest, however, and shown by this table that of the patients reported as having gross or micro scopic endobronchial disease at the time of operation, the postoperative com plication rate was definitely and appreciably increased, 36 to 39 per cent of cases, whereas when no endobronchial disease was seen, the complication rate was a surprisingly uniform average of 26 and 24 per cent

TABLE III RELATIONSHIP OF TUBERCULOUS TRACHEOBRONCHITIS TO THE DEVELOPMENT OF POSTOPERATIVE COMPLICATIONS*

TUBERCULOUS TRACHEOBRONCHITIS	NUMBER DONE	NO COMPLICATIONS NUMBER	ALL COMPLICATIONS		EARLY FISTULA	LATE FISTULA	EMPYEMA	SPREAD OF DISEASE	STUB BORN SPACE
			NO	%					
a									
Gross	50	32	18	36	14	0	14	3	13
None seen	299	220	79	26	47	2	39	25	52
Not stated	251	198	53		28	1	31	23	28
b									
Microscopic	*54*	*33*	*21*	*39*	*15*	*0*	*13*	*5*	*12*
None microscopically	214	162	52	24	31	1	28	12	34
Round cell infiltration	58	44	14	24	11	0	8	3	11
Not stated	274	211	63		32	2	35	31	36
c									
Bronchoscopically seen before	54	44	10	19	6	0	8	2	6
None seen	530	399	131	25	79	2	70	43	81
Not stated	16	7	9		4	1	6	6	6

*Sections *a* *b*, and *c* are not self excluding but show a considerable overlap

When endobronchial disease had been noted bronchoscopically, the compli cation rate was below average (19 per cent) which may well be attributed to the prolonged antimicrobial treatment given for this condition preoperatively

Antimicrobial therapy was first administered at the Sanatorium in the form of streptomycin in February, 1947, followed by PAS in August, 1949, thiosemicarbazone in September, 1950, and isoniazid in June, 1952 During the early years, of necessity, a number of our patients did not receive any anti microbial therapy pre or postoperatively This was followed by a period when patients received streptomycin alone As soon as a second drug became avail able, however, it was our policy to administer at least two of the available drugs in combination at all times even to the point of using thiosemicarbazone more frequently than its therapeutic worth by itself might have warranted

As was to be expected, 52 per cent of all patients who did not receive pre operative *chemotherapy developed one or more of the major complications in* the postoperative period This is shown in Table IV As was anticipated, also, those patients who were able to receive only one drug preoperatively developed fewer complications if the drug was administered for less than three months preoperatively than if given for longer periods, 26 per cent as compared to 50 to 55 per cent. In other words, those patients who received only small quantities of a single drug preoperatively were less likely to develop resistance and so were able to benefit from it for longer periods of time postoperatively. Most of the patients who received a single drug for less than three months did so for approximately three to seven days only. Many of these patients, too, were in a more nearly arrested stage of the disease at the time of operation, being early cases in this resection series. They had reached the so-called "target point" without benefit of antimicrobial therapy.

TABLE IV. COMPLICATIONS RELATED TO THE ADMINISTRATION OF NO PREOPERATIVE ANTIMICROBIAL DRUGS OR A SINGLE DRUG FOR VARIOUS PERIODS OF PREOPERATIVE TIME

	NO PREOPERATIVE DRUGS		ONE DRUG ONLY PREOPERATIVELY													
			<3 MOS.		3-6 MONTHS		6-12 MONTHS		12-18 MONTHS		1½-2 YEARS		2 YEARS +		TOTAL	
	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
Complications	11	52	9	26	6	55	1	50	1	100	0	—	0	—	17	35
No complications	10	48	25	74	5	45	1	50	0	—	1	100	0	—	32	65
Total	21	100	34	100	11	100	2	100	1	100	1	100	0	—	49	100

Table V shows the number and the percentage of patients who developed complications depending upon the type of operation performed and the duration of antimicrobial therapy. The figures for pneumonectomies, lobectomies, and wedge resections would seem to be large enough to be significant.

As was to be expected, where no preoperative chemotherapy was given, the complication rate was high, approximately 53 per cent. It is to be remarked, however, that most of these patients did not have postoperative antimicrobial therapy either.

As therapy with a single drug is no longer necessary or favored, it seemed well to present the picture after the exclusion of the cases which did not receive any antimicrobial drugs or received only one. This is shown in Table VI as it applies to all types of operations.

Actually, there is very little variation in the percentage of patients developing postoperative complications regardless of the shortness or length of time of the preoperative medication. There was a tendency, however, for those patients who received antimicrobial treatment for less than three months to develop more complications than those receiving the drugs from three months to over a year and a half (28 per cent as compared to 19 to 24 per cent). One can only assume that the active component of the disease was not sufficiently controlled in these cases preoperatively.

By the same token, those that received combined therapy preoperatively for a period of over two years had a higher complication rate, also 28 per cent. It is to be remembered, however, that they had received combined therapy for this prolonged period because the seriousness and extent of the disease warranted

TABLE V COMPLICATIONS RELATED TO TYPE OF OPERATION AND LENGTH OF ANTIMICROBIAL THERAPY SHOWN

OPERATION		COMPLICATIONS SHOWN ACCORDING TO LENGTH OF PREOPERATIVE CHEMOTHERAPY															
		NO CHEMO THERAPY		<3 MOS		3 6 MONTHS		6 12 MONTHS		1 1½ YEARS		1½ 2 YEARS		2 YEARS +		TOTAL COMPLICATIONS	
TYPE OF OPERATION	NO	NUMBER	%	NO	%	NO	%	NO	%	NO	%	NO	%	NO	%	NUMBER	%
Pneumonectomy	92	3	60	14	46	8	40	3	30	6	50	3	50	3	33	40	44
Lobectomy	280	5	50	20	23	10	23	17	21	12	29	2	15	3	42	69	25
Bilobectomy	20	3	60	1	14	0	0	1	50	—	—	1	100	0	0	6	30
Lobe + segment	39	—	—	4	25	2	66	5	39	5	83	0	0	—	—	16	41
Lobe + wedge	10	—	—	0	0	0	0	1	25	0	0	—	—	—	—	1	10
Lobe + segment + wedge	2	—	—	1	100	0	0	—	—	—	—	—	—	—	—	1	50
Segmental	48	—	—	5	42	1	25	3	19	2	18	0	0	0	0	31	23
Segment + wedge	3	—	—	—	—	1	50	0	0	—	—	—	—	—	—	1	33
Multiple wedge	9	—	—	0	0	0	0	1	33	1	33	—	—	0	0	2	22
Wedge only	97	0	0	0	0	0	0	3	9	0	0	0	0	0	0	3	3
All cases	600	11	53	45	28	22	23	34	21	26	25	6	19	6	29	150	25

— No cases in group
0 No complications among cases in group

it. In most cases, surgery would not have been either reasonable or possible without this extensive period of preoperative preparation. The total complication rate was 23 per cent for all patients receiving two or more drugs preoperatively.

TABLE VI. COMBINED THERAPY ONLY. COMPLICATIONS RELATED TO DURATION OF TIME OF ANTIMICROBIAL THERAPY PREOPERATIVELY

	<3 MOS.	3-6 MONTHS	6-12 MONTHS	12-18 MONTHS	1½-2 YEARS	2 YEARS +	TOTAL
All cases on combined therapy	128	85	162	103	31	21	530
Number with complications	36	16	33	25	6	6	122
Per cent with complications	28	19	20	24	19	28	23

As deaths reflect to some extent the complication rate, it may be of interest to show (Table VII) that the pneumonectomy operations carried the highest operative and over-all mortality, 8.7 per cent and 20 per cent, respectively. The operative mortality for all cases, 1944 to 1955, was 4.3 per cent within sixty days of operation and 7.8 per cent for all causes from three months to eleven years postoperatively.

TABLE VII. DEATHS, ANY CAUSE, ACCORDING TO TYPE OF OPERATION

TYPE OF OPERATION	NUMBER	OPERATIVE MORTALITY (WITHIN 60 DAYS)	PRESENT STATUS KNOWN	OVER-ALL MORTALITY TO APRIL 30, 1955
Pneumonectomy	92	8 or 8.7%	91	18 or 20%
Lobectomy	280	15 or 5.4%	273	24 or 8.8%
Bilobectomy	20	1	20	1
Lobectomy + segment	39	1	36	1
Segmentectomy	48	1	47	2
Wedge	97	0	96	0
Other	24	0	24	0
All cases	600	26 or 4.3%	587	46 or 7.8%

DISCUSSION

It is generally accepted that, where feasible, a pulmonary resection is the operation of choice for tuberculosis of the lung. In theory, at least, it removes the gross tuberculous lesion from the body. Unfortunately, unsuspected or microscopic areas of tuberculous disease are frequently left behind either within the lung parenchyma or in the bronchial walls.

It is suggested by the present analysis that men do not respond to this form of surgery as well as women and that, after the age of 40 years, the response becomes less favorable with the passage of time. Of the 15 men over the age of 40 years who had evidence of gross or microscopic endobronchial disease and underwent a pneumonectomy, or a lobectomy, or a lobectomy in combination with a more minor operation, 66 per cent developed complications. Of the remaining 49 men over 40 years of age without gross or microscopic endobronchial disease, 45 per cent developed complications compared to an average of 25 per cent for the whole group of resection cases. (Of the last 400 cases of this series, the complication rate was only 19.2 per cent, all having received combined therapy) The presence of previous or concurrent tuberculous endobronchial disease, known or unsuspected, increases the hazard of the operation, especially if the bronchial lesion occurs at the site of resection It has been shown previously that ulcerative lesions of the bronchus will heal rapidly, often within one to three months,[1] following the initiation of antimicrobial therapy Unfortunately, we lack similar knowledge regarding the rate of healing of submucosal tuberculous lesions which still present an operative hazard

It would seem from the figures presented that there is some merit in trying to bring the patient to the so called "target point" before undertaking surgery if by "target point" we may indicate relative stability of the lesion obtained by treating the patient with antimicrobial drugs sufficiently long to arrest submucosal tuberculosis as well as eliminate the exudative components of the parenchymal lesion Less than three to six months antimicrobial therapy would seem to be inadequate Three to eighteen months preoperative "combined drug" therapy, where indicated, would not seem to increase the risk of developing complications during the postoperative period

Many of those patients who received drug therapy for over two years before operation did so because the disease was too extensive or too active to warrant resection sooner The prolonged drug therapy brought them to a state of improvement in which surgery could be undertaken with relative safety The prolonged period of preoperative treatment should not be blamed for causing postoperative complications In many cases it was lifesaving and in 72 per cent of those who received drugs for over two years there were no complications

The operative death rate in this series was 4 3 per cent with an over all rate of 7 8 per cent Excluding the pneumonectomies, the rate for the series is 3 5 per cent and 5 6 per cent, respectively, with no mortality for wedge resections Undoubtedly, a pneumonectomy is the resection operation of the greatest magnitude Not only does it carry with it the greatest surgical risk, but the patient is like a two engine airplane carrying on with only one motor for the rest of his life If anything, even trivial, happens to the remaining organ, the result may be catastrophic Similarly, a single fuel pump, the heart, carries the responsibility for circulating the required vital fluid of the whole mechanism to the single motor Even a minor failure here in the circulating mechanism will diminish its function below a life sustaining level There is an impression that cor pulmonale plays a larger part in our mortality figures than was at one time thought to be the case

SUMMARY

1 Six hundred consecutive cases of pulmonary resection at the Nova Scotia Sanatorium have been reviewed

2 The possible influence of age, sex, endobronchial tuberculosis, and the duration of preoperative antimicrobial therapy have been tabulated

3 Mortality figures are given

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INTRATHORACIC NEUROFIBROMA OF THE VAGUS NERVE

A Case Report

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NEUROFIBROMAS arising in the posterior mediastinum are not uncommon, and at times they are associated without generalized neurofibromatosis (von Recklinghausen's disease). Such posterior mediastinal tumors have their origin from the intercostal nerves or the sympathetic chain. Neurofibromas of the cranial nerves are not common, the commonest nerve involved being the acoustic nerve.[1] Tumors of the cervical segment of the vagus nerve have been reported by a number of authors, but those arising in the thoracic segment are rare. Blades and Dugan[2] reported the first patient suffering from a thoracic neurofibroma of the left vagus nerve and its successful extirpation. Furrer and Fox[3] reported a patient with a perineural fibrosarcoma of the left vagus nerve which was discovered at post-mortem examination. Efskind and Liavaag[4] in their review of 21 cases of intrathoracic neurogenic tumors reported 1 patient with a neurinoma which had its origin in the recurrent laryngeal nerve. Recently Gerbode and Marguiles[5] reviewed the literature on this subject and reported 1 case of neurofibromatosis with neurofibromas of vagus nerve in the superior mediastinum associated with pectus excavatum. Since then Gilbertsen and Lillehei[6] have reported the instance of a patient with bilateral neurofibroma of the vagus nerve unassociated with generalized neurofibromatosis. It would appear that neurofibromas of the vagus nerve associated with generalized neurofibromatosis are uncommon. The present case report falls within this category. The tumor arose from the left vagus nerve immediately below the origin of the recurrent laryngeal nerve and was successfully extirpated.

CASE REPORT

S. W. (A-169238), a 30-year-old white man, was admitted to the Harper Hospital, Detroit, Mich., on Dec. 29, 1953, because of a mediastinal mass which was discovered on a routine yearly chest film. The patient was an employee of a public school system, and had had chest x-rays each year. He stated these chest examinations had been negative. The patient had no complaints or symptoms. There was no family history of tuberculosis, but the patient stated that he had had a positive tuberculin test for years. The past history revealed that he had had a toxic goiter as a boy, which was treated medically and later by x-ray. An appendectomy had been performed ten years before.

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On admission the physical examination revealed a well developed, well nourished, white man, appearing healthy, and in no apparent distress The physical examination was entirely negative, and there was no evidence of any neurofibromatosis The blood pressure was 140/90 The pulse was 74 per minute The admission complete blood count, urine analysis, total serum proteins, and albumin globulin ratio were within normal limits

An x ray of the chest revealed an oval soft tissue mass in the left mid mediastinal area just above the left hilum Fluoroscopically, the mass lay just above the main pulmonary artery on the left. No pulsations were observed in this area The nature of this mass could not be determined by roentgenographic studies.

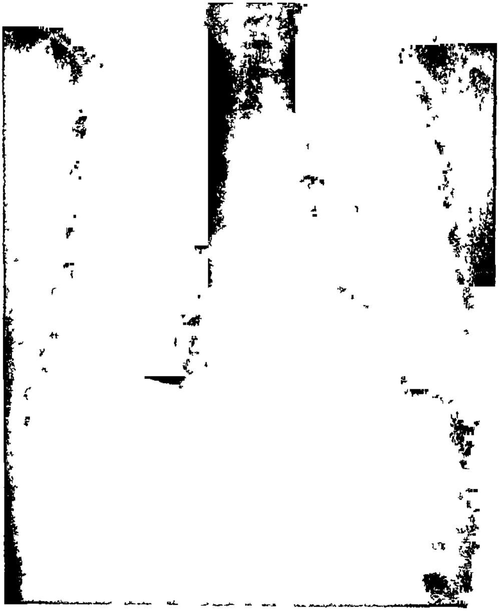

Fig 1—A posteroanterior projection of the chest reveals a tumor mass protruding from below the arch of the aorta, the nature of which was not determined until at the time of operation

On Dec 30, 1953, the patient was taken to the operating room and under intravenous Pentothal, curare, procaine, and intratracheal nitrous oxide and oxygen anesthesia with the patient in the left lateral position, an exploratory thoracotomy was performed through the bed of the fifth rib Exploration of the mediastinum revealed a firm, smooth, oval shaped tumor mass present just above the left main pulmonary artery and below the aortic arch Further dissection revealed that the left vagus nerve entered the tumor mass and emerged from it The recurrent laryngeal nerve arose approximately 5 mm above the tumor At the distal end of the tumor was the remaining normal trunk of the vagus nerve and the pulmonary plexus The tumor was removed amputating the vagus nerve superiorly just below the recurrent laryngeal

The patient tolerated the procedure well, and no evidence of rise of pulse rate or drop in blood pressure was seen at any time. There was no evidence of laryngeal paralysis. Postoperative examination of the larynx revealed both cords of equal amplitude.

Pathology Report.—Gross Examination: The specimen was a piece of tissue 5 by 2 by 2 cm. which was pink, smooth, and at one point, contained a firm 0.5 cm. nodule. The tumor appeared cystic. On cut surface the specimen was gelatinous.

Microscopic Examination: Neurofibroma of the vagus nerve.

The patient had an uneventful postoperative course and was discharged from the hospital on Jan. 8, 1954.

SUMMARY

A case of neurofibroma of vagus nerve in the chest unassociated with generalized neurofibromatosis is presented. The tumor was successfully removed.

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VENTRICULAR FIBRILLATION IN ARTIFICIAL HYPOTHERMIA

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ARTIFICIAL hypothermia is now increasingly attracting the attention of physicians and surgeons for its many applications in several fields. In its use as a valuable tool in cardiac surgery, however, the surgeon has been faced with the problem of ventricular fibrillation that seems to occur more readily than at normal temperatures. Bailey and associates[1] have reported ventricular fibrillation as occurring in 66 per cent of their cases; other authors have similar figures.[2]

There is not a thorough agreement on the mechanism of ventricular fibrillation. There are, however, some well established facts. One condition for the appearance of fibrillation is heterogeneity in the properties of the ventricular muscle.[3,4] Another condition is the initiation of an impulse at the end of the functional refractory period of the preceding one.[4-7] It was decided, therefore, to apply these known facts to the experimental study of this cardiac disorder under hypothermia with the idea of finding some practical applications which might help in solving this important problem.

METHOD

Rats, under Nembutal anesthesia (50 mg. per kilogram, intraperitoneally), were used. A tracheal cannula was inserted and artificial respiration instituted, the chest was opened through removal of the sternum and the costal cartilages, leaving the heart widely exposed. The opened pericardial sac was sutured to the skin. On the ventral surface of the ventricle were placed electrodes for stimulation and recording (small steel serrefines fixed to the epicardium and hanging from fine flexible wires). The animal was then placed in a small plastic box, closed except for some openings for the artificial respiration tube, the electrodes, and the thermometer (which was a large cuvette mercury type placed inside the abdominal cavity through a small opening in the anterior wall).

The properties of the ventricular muscle studied were: (*a*) the threshold voltage to electrical stimulation with square pulses of 1 millisecond duration and at a frequency just above the sinusal rhythm, (*b*) the functional refractory period, for example, the shortest interval between two propagated responses to two shocks of 150 per cent threshold voltage, or the maximal frequency at which the ventricle could be driven with a short train of pulses of that magnitude, (*c*) the conduction velocity of the impulses, judged by the latencies for shocks of that same intensity and of frequency just above the actual heart

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rate; (*d*) the voltage amplitude of the electrical responses recorded from any point on the surface of the ventricle to an indifferent electrode placed on the neck of the animal and grounded; and (*e*) the rate of rise of the ascending limb of the monophasic action potential.

Once the basal values of these properties were obtained, the box was surrounded by cracked ice and several measurements of those properties were made during the cooling period. The records were photographed from a cathode ray oscillograph (DuMont 322) using their own D.C. coupled amplifier. In some cases the records were taken on a three-channel inkwriter galvanometer (Model III D Grass electroencephalograph). In this last case, the action potentials were fed through a Grass Converter-Demodulator and re-

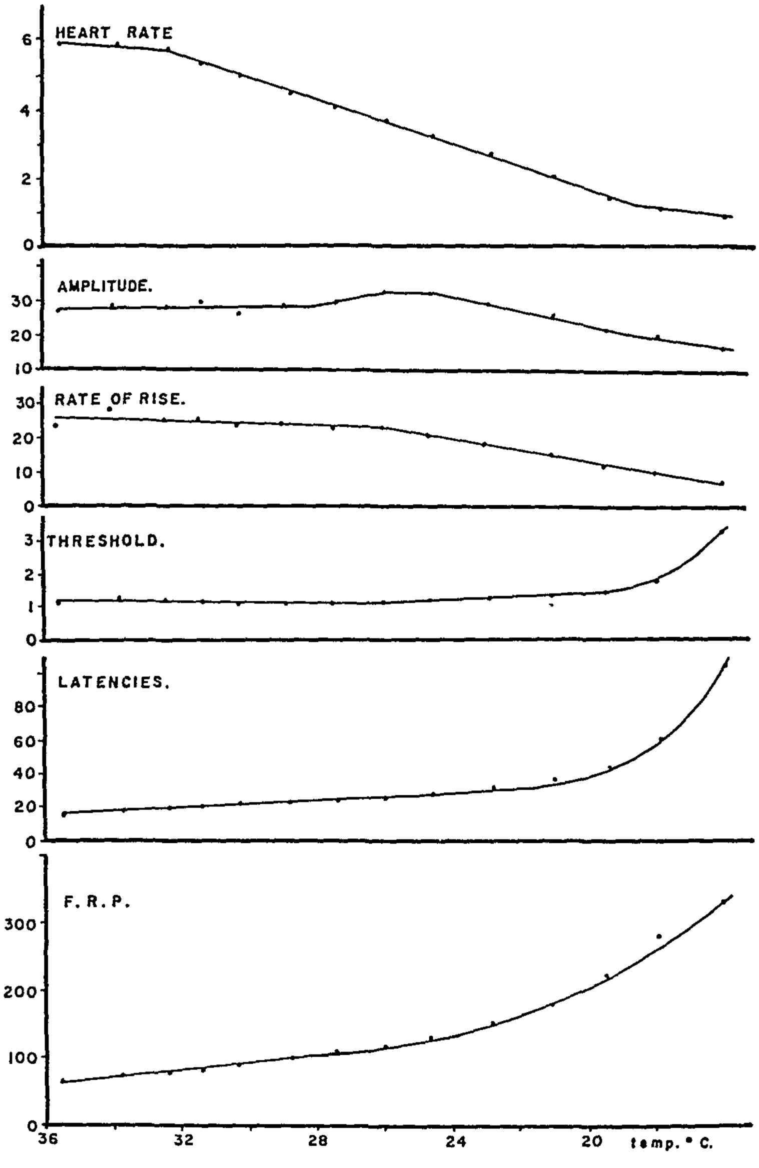

Fig. 1.—Curves from one experiment showing the changes in some ventricular properties during gradual cooling. Ordinates from above to below: Heart rate, in beats per second. Amplitude of the monophasic action potential in millivolts. Rate of rise of the ascending limb of the monophasic action potential, arbitrary units. Threshold to electrical stimulation, in volts. Latencies of the stimulated responses, in milliseconds. Functional refractory period, in milliseconds. Common abscissas: Temperature in degrees centigrade.

corded with a low frequency galvanometer coil in order to have accurate records due to the infinite time constant of this circuit The stimuli applied came from a model S4B Grass stimulator through a Grass stimulus isolation unit

RESULTS

A *Normal Beats*—The heart rate changes linearly with the temperature The thermal coefficient (Q_{10}) has an average value of 2 39, the lowest figure found was 1 77 and the highest one was 3 07

The voltage amplitude of the action potential practically does not change until a critical point, about 25° C Below this value, the action potential begins to decline rapidly Sometimes before that point in the cooling process is reached there is a gradual increase of the amplitude which can be as much as 10 to 20 per cent of the control values

The changes in the rate of rise of the ascending limb of the monophasic action potential follows a parallel course with the changes in amplitude (Fig 1)

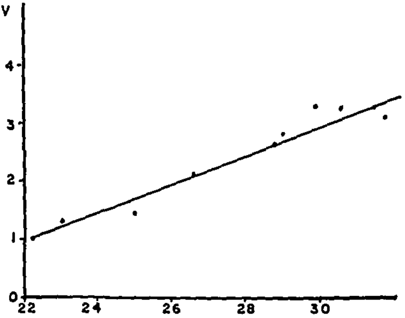

Fig 2—Relation between conduction velocity of impulses (reciprocals of the latencies in milliseconds × 10 in ordinates) and rate of rise of the ascending limb of the monophasic action potential (arbitrary units in abscissas)

B *Responses to One or Two Stimuli*—The threshold voltage either does not change at first or presents a gradual decrease of small magnitude until a temperature of about 20° C at which it tends to rise rapidly (Fig 1)

The latencies of the responses increase in an exponential like manner The changes are very small for temperatures above 25° C, usually not reaching values greater than 25 per cent of the control even at this particular temperature After that point a slight reduction in temperature results in a great increase in latency (Fig 1)

There is a linear relation between conduction velocity of impulses and the rate of rise of the ascending limb of the action potential (Fig 2)

The functional refractory period follows a parallel change, although the increase is always greater than the corresponding variation in latency At 25° C, it is usual to record an increase of 50 per cent, and sometimes even 100 per cent above the control values

C *Responses to Trains of Stimuli*—The responses to trains of stimuli, of 1 second duration, at different frequencies, and of intensity of 150 to 200 per cent threshold, were recorded. Successive latencies and amplitudes of the responses were measured.

During the control conditions the amplitude of the responses are more or less the same for the different frequencies of stimulation. At higher frequencies (12 to 16 per second), there is a gradual fall in amplitude for the successive responses until suddenly the ventricle begins to alternate to the shocks. The latencies shortened in the successive responses to relatively low frequencies and showed a moderate increase with the higher ones. With the highest ones, there is some alternation until finally an impulse is missing (Fig. 4).

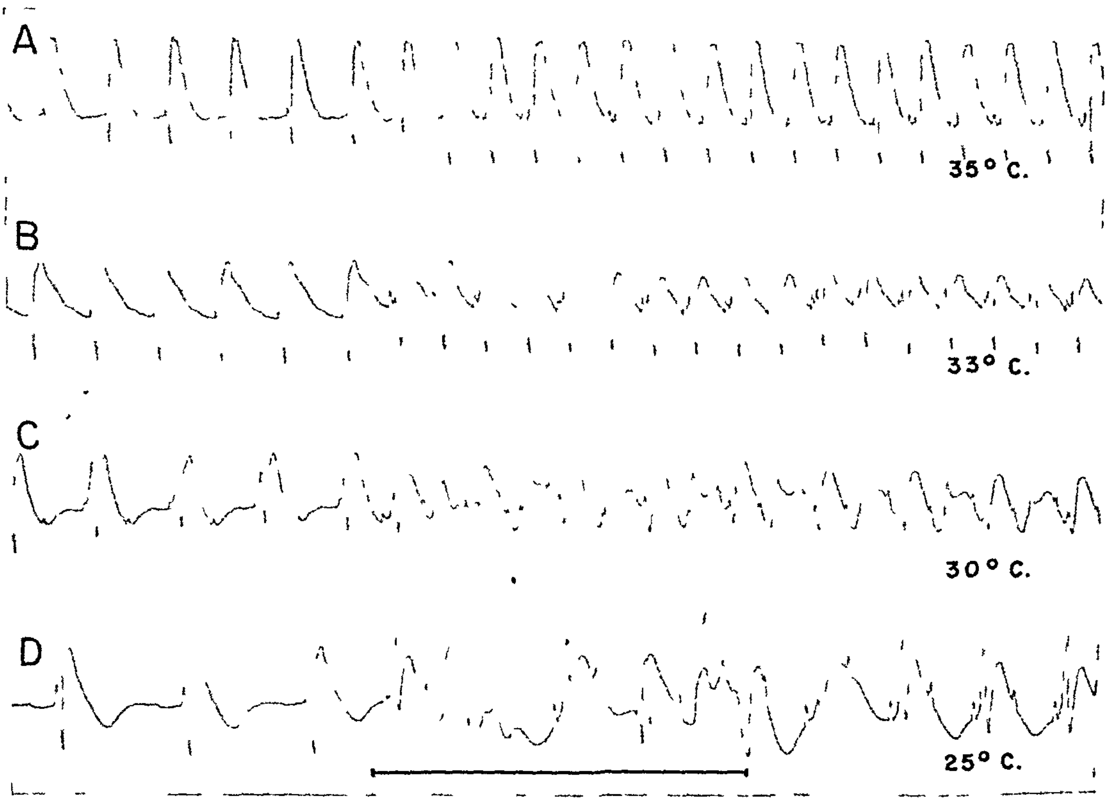

Fig. 3.—Gradual development of heterogenicity in the ventricular muscle with falling of the temperature The irregularities in the responses show that speed of conduction and refractoriness are not the same for all active fibers. Electrograms taken with the electroencephalograph, stimulating and recording electrodes over the ventricle Frequency of stimulation 9 per second, the horizontal line below represents one second

As the cooling advances the amplitudes become irregular, the more so the lower the temperature. Fig. 3 gives some examples for a frequency of stimulation of 9 per second. The latencies increase markedly and show the alternation sooner, as illustrated in Fig. 4, for a frequency of 8 per second. The action potentials for the longer latencies are the smaller ones. Sometimes the action potential is so small that it looks just like an irregular base line.

D. *The Effect of Aconitine.*—Aconitine crystals applied locally over the surface of the ventricle do not give rise to the development of an ectopic foci of impulses at the lower temperatures, even if the heart is made slower by cutting the artificial respiration, or after a train of electrical shocks at relatively high frequency, conditions which always make this kind of ectopic foci to discharge in a heart at normal temperature.

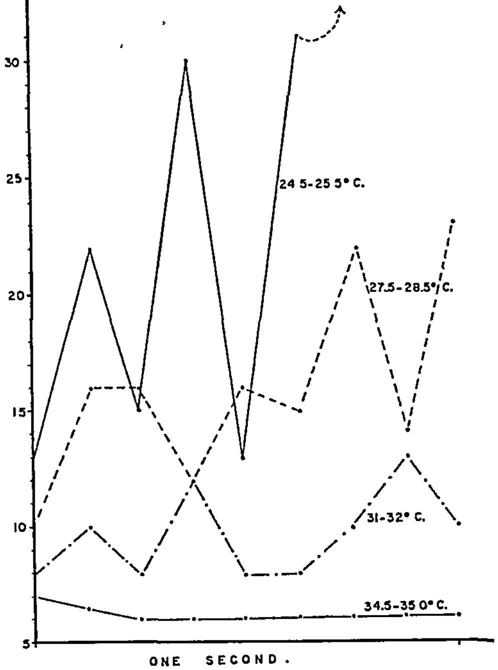

Fig. 1.—Latencies for the individual responses to each of a train of electrical shocks at a frequency of 8 per second and at the temperatures given for every curve. Ordinates: latencies in milliseconds. Abscissas: time (each point is plotted at ⅛ second interval).

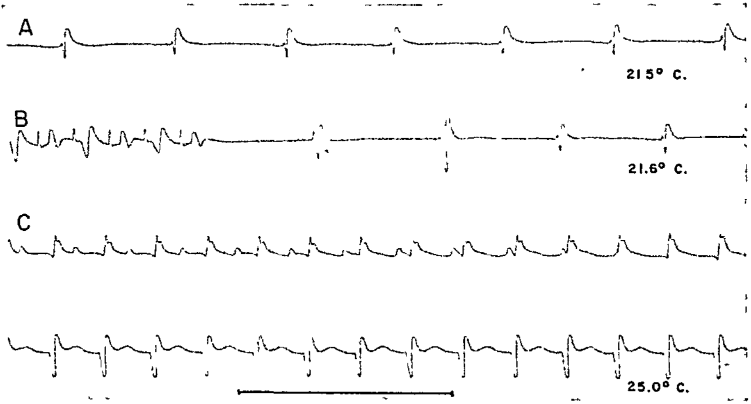

Fig. 5.—Effect of aconitine locally applied on a cool ventricle. Electrograms of the heart: *A*, Normal ventricular beats, in spite of the fact that the aconitine have been already applied. *B*, After starting the warming up. There is some extrasystolic activity but only after a train of electrical shocks at a frequency of 6 per second. *C*, At a higher temperature an ectopic focus begins to discharge. Here, the upper record shows the auricular activity at a lower rate and was taken from an electrode on the right appendix to another point on the ventricle. Time calibration: one second.

When the animal is warmed again, the aconitine applied may have the usual effect (Fig. 5).

DISCUSSION

Some of the results can be analyzed from the pure physiologic point of view. The amplitude of the monophasic action potential, the rate of rise of the ascending limb, and the threshold to electrical stimulation vary in a manner similar to that described for the nerve fibers.[5, 6]

For the increase in height of the action potential, no satisfactory explanation has been advanced. For the decline of those properties it is interesting to recall that Lorente de No[6] pointed out that "the effect of a lowering of the temperature closely resembles that which is produced by lack of oxygen," and that "the changes in the spike potential are referable mainly to a reduction of the rate of those chemical reactions which underlie the alteration itself and the restoration of the membrane potential."

The relation between speed of conduction and rate of rise of the ascending limb of the monophasic action potential is in agreement with the observations of Gasser[5] on the nerve fibers, and also with the local circuit theory for the propagation of impulses.

Let us now examine the data in regard to the mechanism of ventricular fibrillation. According to the circus movement theory to explain this cardiac disorder there is required a closed path of such an extension as to contain an impulse. The wave length of this impulse must then be smaller than the path itself. The heart of the large animals always has the proper dimensions to enclose one or several of those closed paths. Any condition which can shorten the wave length, for example, increasing the speed of conduction, or shortening of the refractory period, or both, will favor the appearance of fibrillation. The enlargement of the possible paths to a traveling impulse will act in a similar way, as does the establishment of heterogenicity in the properties of the individual muscle fibers. All that is necessary in such conditions is to initiate a unidirectional wave of impulse by applying a stimulus at the end of the functional refractory period of a preceding one.[7]

When this study was started it was expected to find, with the lowering of the temperature, a decrease in speed of conduction with either no change in duration of the refractory period or at least with a small increase in this period, but not in proportion with the change in conduction velocity. As the data show, the change in the refractory period, as this is currently determined, was greater than the increase in latency. This result would be against the development of fibrillation, as the wave length of the impulses is now longer.

The results with trains of stimuli showed, however, that latencies do increase markedly at successive responses (Fig. 4). Still more, the heterogenicity in the individual muscle fibers become more evident the lower the temperature is (Fig. 3). This means that the wave length progressively shortens at the same time that the path enlarges by making itself irregular. Thus, the present results are compatible with the circus movement theory as the mechanism for ventricular fibrillation. Even if the refractory period will have shown an increase much greater in proportion to the degree in which conduction velocity was slowed, the simple development of heterogenicity in the muscle fibers alone, as revealed for the variability in latencies and the appearance of irregularities in the amplitude and configuration of the responses, will be enough to make the ventricle apt to develop fibrillation and of a less easily reversible kind, as is the one found in the clinical observations.

The fact that the threshold of stimulation does not change markedly with the lowering of temperature makes reasonable the possibility of the initiation of an impulse which, under proper conditions, can induce fibrillation.

This type of result shows that sometimes it is not enough to make measurements at static conditions. The two shock method for determining the length of the refractory period and the latency to the first shock were giving us a wrong picture. The observations with trains of shocks, fortunately, gave us a definite answer.

The studies made by Sherf[8] with aconitine led him to believe that multiple ectopic foci were responsible for ventricular fibrillation, the smaller waves seen in the records taken from a fibrillating heart would be local nonpropagated responses. Garcia Ramos and Miledi[4] have shown, however, in a study with microelectrodes, that there are no such local potentials, every action potential recorded is a propagated one, and the frequency of activity is just limited by the functional refractory period of the individual muscle fibers at that moment. The irregularities in the records are due to the lack of synchronization of the different active elements.

The present observations with aconitine are not a direct disproof of the multiple foci theory. In fact, the effects of this drug cannot be used in support of any theory. The conditions of its action are artificial. The only thing one can say is that the lack of effect of aconitine at lower temperatures, which was described precisely by Sherf,[8] makes all the arguments based on its action unsuitable for the explanation of the mechanism of ventricular fibrillation.

On the other hand, there is not any evidence that the potential automatic activity of the ventricular muscle is as good at low temperature as it is at the normal one.

The clinical observations of Bigelow and co-workers that maintenance of blood pressure above 80 mm. Hg and hyperventilation of the patients give some protection against the ventricular fibrillation of hypothermia suggest that hypoxia of the muscle can be a factor. It is true that at low temperatures the oxygen consumption of the heart is greatly diminished and that the fall in blood pressure could be not serious enough to interfere with the proper irrigation of the muscle. If one takes into account, however, that aortic pressure is not the only factor in coronary circulation and that the energy of the contractions and the heart rate have also a direct effect on improving it, as shown by García Ramos, Alanis, and Rosenblueth[9] in the isolated, perfused dog's heart, we have a greater support for this idea. In clinical as well as in the experimental observations, it is found that the heart rate decreases markedly, and Bigelow and co-workers[2] found that the cardiac output shows a progressive fall during cooling.

It was pointed out previously that the changes in the electrical events of heart activity can possibly be ascribed to a failure of the oxygen utilization mechanisms.

There is another observation that favors the idea of hypoxia of the heart muscle at low temperatures. Swan[10] has reported that acetylcholine and Prostigmin seems to protect the heart against fibrillation, and García Ramos and Ramírez de Arellano[11] showed that acetylcholine depresses the oxygen consumption of frog's and turtle's hearts.

An analysis of the conditions for establishment of heterogenicity in the properties of ventricular muscle fibers due to hypoxia permits one to state some practical measures as follows: (*a*) try to maintain the coronary blood pressure as high as possible; (*b*) keep the arterial oxygen saturation at normal values; and (*c*) reduce the sympathetic activity, the local as well as the general. Adrenalin is now a well-known hypoxiating agent.[12] The same can be said about some other sympathomimetic amines. All these measures will tend to reduce the heterogenicity of the heart muscle and so give some protection against fibrillation.

SUMMARY

An experimental study on rats under artificial hypothermia shows that lowering the temperature of the animal increases the heterogenicity of the ventricular muscle making it more apt to present ventricular fibrillation. This change is probably produced by the depression of oxidative reactions and by the reduction in the proper irrigation of the heart. These two factors together interfere with the processes of restoration after activity.

A discussion is made on the physiologic significance of the changes with respect to the mechanism of ventricular fibrillation. The data are in favor of the idea of a circus movement more than that of multiple ectopic foci.

Some practical applications are derived from the analysis of the results and of the clinical observations. Reduction of extra oxygen consumption and an adequate oxygenation of the heart muscle will tend to protect it against fibrillation.

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THE ACUTE EFFECTS OF RESECTION OF PULMONARY TISSUE ON SOME PULMONARY FUNCTIONS IN THE DOG

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EVALUATION of the acute response of anesthetized dogs to resection of pulmonary tissue and to reduction of the blood supply to the lung has been confined largely to hemodynamic studies. Long and associates[1] found that pneumonectomy caused a 2 to 13 mm. water rise of the mean pressure in the right ventricle and a variable effect on the cardiac output. Fineberg and Wiggers[2] found that failure of the right ventricle developed only after approximately 60 per cent of the pulmonary artery was occluded. Gibbon and Churchill[3] found that occlusion of approximately 60 per cent of the cross-sectional area of the pulmonary artery produced a fall of systemic blood pressure whereas the systemic pressure remained constant or was elevated after occlusion of from 40 to 73 per cent of the lobar branches of the pulmonary artery.

This report concerns the immediate effects of resection of the left lung and of the upper and middle lobes of the right lung on alveolar and arterial blood gas tensions, the mean pressure in the pulmonary artery and the systemic arteriovenous oxygen difference in 6 dogs. These data were obtained in order to assess the effect of resection of large amounts of pulmonary tissue on various aspects of pulmonary function of the remaining lung and to obtain information as to the minimum amount of pulmonary tissue that must be preserved in order to carry on these functions.

METHODS

Dogs were anesthetized by the intravenous injection of Pentothal (25 mg. per kilogram) followed by barbital sodium (200 mg. per kilogram). The dogs were ventilated through a tracheotomy tube by means of a constant volume Bird respirator pump. This pump permitted collection of the expired air and substitution of a low oxygen mixture for room air as the inspiratory gas. Pump rate and stroke volume were kept constant throughout the experimental period. The lungs were kept inflated throughout the experiment by passing the expired air through a column of 5 cm. of water. The femoral artery was cannulated for the collection of arterial blood samples. The chest was opened in the fourth to the fifth intercostal space and the sternum was transected. A polyethylene

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cannula was inserted through the wall of the right ventricle (through a 17-gauge needle) into the pulmonary artery for measurement of the mean pressure in the pulmonary artery and for collection of mixed venous blood. The pressure at the level of the catheter tip was measured at the end of each experiment and was used as the zero pressure. After a 20-minute period of ventilation with room air, expired air was collected for three minutes. Samples of arterial and of mixed venous blood were drawn simultaneously at a constant rate throughout this 3-minute period. Twelve per cent oxygen was then administered as the inspiratory gas and, after twenty minutes, the air and blood samples were collected again. Ventilation was then re-established with room air, and the roots of the left lung, right upper and middle lobes were ligated and clamped and the pulmonary tissue distal to the clamps was resected. After a 20-minute interval, expired air and blood samples were obtained and the dog was placed on 12 per cent oxygen. Twenty minutes later the final expired air and blood samples were collected.

Arterial blood gas tensions were measured immediately by the direct technique developed by Riley and his co-workers.[4] The equilibrating water bath was kept 0.4° C. lower than the dog's rectal temperature, approximately the temperature existing in the lung.[5] Expired gas volumes were measured in a Tissot spirometer and corrected to 38° C. [BTPS (body temperature pressure, saturated)] for calculation of ventilatory volumes and to STPD (standard temperature pressure, dry) for calculation of oxygen consumption. Expired gas composition was measured on the Scholander microgas analyser[6] and the respiratory quotient was calculated. The alveolar oxygen tension was calculated from the alveolar equation,[7] substituting arterial for alveolar carbon dioxide tension. The lung dead space was calculated from the Bohr equation. The mean pressure in the pulmonary artery was measured on a saline manometer. The arterial and venous oxygen saturations were measured by a spectrophotometric technique.[8]

RESULTS

Data obtained from the 6 of 7 animals which survived throughout the experimental period are presented in Table I. In each animal the arterial carbon dioxide tension, both during the inspiration of room air and of 12 per cent oxygen, was lower after resection of the pulmonary tissue than it had been during the control period. Since the expired carbon dioxide tension was virtually unaffected by pneumonectomy the lung dead space was always lower after resection [83 to 95 per cent (average: 86 per cent) of the control during room-air breathing and 82 to 94 per cent (average: 88 per cent) during 12 per cent oxygen breathing]. The arterial oxygen tension measured during inspiration of room air increased in 4 of the dogs after resection. In all but 1 animal the alveolar oxygen tension rose. The alveolar-arterial oxygen tension difference did not change significantly. During the inspiration of 12 per cent oxygen the arterial oxygen tension was higher in 5 of the animals after resection, the alveolar oxygen tension was higher in all of the dogs, and the alveolar-arterial oxygen tension difference was not altered significantly.

TABLE I

DOG NUMBER	INSPIRED OXYGEN TENSION (MM HG) $P_{I_{O_2}}$	EXPIRED CARBON DIOXIDE TENSION (MM HG) $P_{E_{CO_2}}$	RESPIRATORY QUOTIENT R	ARTERIAL CARBON DIOXIDE TENSION (MM HG) Pa_{CO_2}	ARTERIAL OXYGEN TENSION (MM HG) Pa_{O_2}	ARTERIAL OXYGEN SATURATION (PER CENT) Sa_{O_2}	VENOUS OXYGEN SATURATION (PER CENT) $S\bar{v}_{O_2}$	HEMATOCRIT HCT	DEAD SPACE C C (BTPS) V_D	OXYGEN CONSUMPTION C C/MIN (STPD) V_{O_2}	PULMONARY ARTERY PRESSURE (CM SALINE) PAP	EFFECTIVE ALVEOLAR OXYGEN TENSION (MM HG) $P^{\circ}_{A_{O_2}}$
1	145	10.3	0.81	33	87	93	70	48	98	86	22	106
	85	8.5	0.94	31	51	80	42	48	103	63	21	52
	145	10.7	0.87	31	82	92	51	53	84	82	29	110
	85	8.8	1.03	30	56	82	44	54	95	56	27	56
2	148	13.1	0.80	53	87	92	64	57	64	79	27	84
	87	13.2	0.85	57	34	48	28	60	66	75	36	22
	148	12.9	0.80	47	85	92	62	61	55	75	39	92
	87	12.9	0.91	56	29	37	20	64	62	66	51	25
3	147	10.1	0.76	35	95	90	58	37	127	104	18	103
	86	9.9	0.84	36	50	86	40	40	130	93	30	44
	147	11.1	0.78	31	96	93	49	44	105	108	27	109
	86	10.0	0.88	31	55	80	40	44	115	88	30	51
4	148	11.8	0.70	60	70	75	67	52	63	76	29	68
	87	8.7	0.97	66	16	15	10	57	72	45	40	12
	148	12.6	0.75	58	80	83	76	53	53	71	37	75
	87	9.8	0.97	58	28	20	11	53	61	42	44	27
5	149	12.5	0.86	42	90	92	45	36	76	86	21	102
	87	13.2	0.93	43	37	39	11	38	80	84	36	41
	149	12.6	0.85	41	96	85	25	38	63	95	33	102
	87	12.6	0.98	41	40	35	6	35	64	83	47	45
6	149	15.3	0.81	51	84	85	73	46	64	99	30	89
	87	14.2	0.93	48	38	49	35	46	65	80	31	36
	149	13.0	0.84	44	91	90	61	44	61	79	38	98
	87	13.2	1.05	38	55	76	50	45	53	64	39	51

Data on 6 dogs before and after resection of the left lung and the right upper and middle lobes of the right lung. In each dog the first two lines represent control observations first during inspiration of room air then of 12 per cent oxygen and the second two lines are the data obtained after resection first during inspiration of room air then of 12 per cent oxygen

During room-air breathing the mean pressure in the pulmonary artery was always higher (7 to 12 cm. saline) twenty minutes after resection of lung tissue than it had been during the control period. The oxygen consumption was approximately unchanged before and after resection in 5 of the dogs, and in Dog 6 it fell. Since the arterial-venous oxygen difference was larger after resection in all but 1 dog, the cardiac output was diminished (33 to 89 per cent) in all except one experiment. The calculated pulmonary vascular resistance (pulmonary artery pressure/cardiac output) was increased in each instance (120 to 385 per cent of the control, average: 215 per cent). During the inspiration of 12 per cent oxygen the pressure in the pulmonary artery was, with one exception, elevated after resection and the calculated resistance to flow through the lung was always increased.

DISCUSSION

These experiments concerned the effects of resection of a large percentage of the lung (according to data of Rahn and Ross,[9] approximately 68 per cent of the total lung). Six dogs survived the procedure, and the seventh dog died two minutes later. All the surviving dogs showed no impairment of the pulmonary functions studied. Although the pressure in the pulmonary artery increased, cardiac output was sufficient to maintain the oxygen consumption at the control level in 5 of the dogs. Since pulmonary ventilation remained constant, and since the dead space was decreased after resection, the arterial carbon dioxide tension fell while the alveolar oxygen tension rose. The alveolar-arterial oxygen tension difference was not significantly increased, except for Dog 1 in which the tension difference during room-air breathing increased 9 mm. Hg. In the other dogs, the changes were within the limits of experimental error (7 mm. Hg). Since this tension difference is a reflection of different phenomena during the inspiration of room air and during the inspiration of a low-oxygen mixture, two inferences may be drawn from these data. The tension difference during room-air breathing is due to the presence of venous admixture to arterialized blood and to the presence of different ventilation-perfusion ratios in different parts of the lung.[10, 11] Since the tension difference did not increase after resection of 68 per cent of the pulmonary tissue, pulmonary ventilation–perfusion relationships in the remaining lung were apparently unchanged. The absence of a significant tension difference during low oxygen breathing after resection of 68 per cent of the pulmonary tissue indicates that the diffusing capacity of the lung was so high that no measurable difference existed between the oxygen tension in the alveoli and in the blood leaving the alveoli. In these dogs, the average arterial carbon dioxide tension after resection, during 12 per cent oxygen breathing, was 44 mm. Hg and the average effective alveolar oxygen tension was 43 mm. Hg, an insignificant difference. Since the average arterial-venous difference was 27 per cent saturation, the difference between the alveolar and the mean capillary oxygen tension was 8 mm. Hg or less.[12] The average oxygen consumption was 67 c.c. per minute, so the oxygen diffusing capacity averaged 8 c.c. per minute per millimeter of mercury or greater. Since only 32 per cent of the lung tissue was present, the diffusing capacity of the entire lung would have been at least 25 c.c. per minute per millimeter of mercury.

Although resection of pulmonary tissue seemed to have no discernible effect on pulmonary blood-gas relationships, there was an obvious increase in the pulmonary vascular resistance. From the data in Table I the resistance during room-air breathing increased to 120 to 385 per cent of the control (average: 217 per cent) and during 12 per cent oxygen breathing 129 to 386 per cent (average: 215 per cent). This was not as great as one might expect on mechanical grounds alone when 68 per cent of the vascular bed was removed. A previous study of the perfused lung of the dog has revealed that a high intravascular pressure serves to increase the size of the pulmonary vascular bed and thereby decrease pulmonary vascular resistance.[13] Thus, the calculated resistance is a function of the pressure in the pulmonary artery, and one cannot relate the resistance to the amount of lung tissue present unless the pressure remains constant.

The effect of further reduction of the pulmonary vascular bed was investigated in 5 of these dogs. In each instance, ligation of the right lower lobe, leaving approximately 8 per cent of the lung in place,[9] resulted in extreme pulmonary hypertension (59 to 88 cm. saline), followed, in five to thirty-five minutes, by a fall of the pressure in the pulmonary artery, dilatation of the right ventricle, and death. In 4 of these animals marked arterial unsaturation developed during the period of pulmonary hypertension (25 to 36 per cent in 4 dogs, 88 per cent in the fifth) and was completely overcome, in the two instances studied, by the inspiration of oxygen. Such a marked effect of oxygen indicates that the reduction of the arterial oxygen saturation was largely due to a diminished diffusing capacity of the lung so that a large difference existed between the oxygen tension in the alveoli and in the blood leaving the alveoli. Inspiration of oxygen raised the alveolar oxygen tension to some 700 mm. Hg and increased the rate of diffusion of oxygen across the alveolar membrane into the blood. A similar phenomenon occurs after diffuse embolization of the lung.[14] The reduction of diffusing capacity after resection of 92 per cent of the lung may be attributed to the removal of diffusion surface and to reduction of alveolar perfusion of the remaining lung resulting from overdistention of the alveoli by the undiminished ventilatory volume which was necessary for maintenance of adequate alveolar ventilation. The close relationship between the circulatory and diffusion functions of the lung is indicated by the fact that both were relatively unimpaired after resection of 68 per cent of the pulmonary tissue whereas right heart failure and limitation of diffusion of oxygen both became apparent after resection of 92 per cent of the lung tissue. The pulmonary vascular reserve was sufficient to maintain adequate circulation and diffusion of oxygen when 68 per cent of the lung was removed, but 8 per cent of the lung contained insufficient vascular bed to maintain either of these functions.

SUMMARY

Acute resection of approximately 68 per cent of the pulmonary tissue has been performed in 7 dogs. One dog died. The remaining dogs showed an increase of the pressure in the pulmonary artery, a small decrease in the arterial carbon dioxide tension and lung dead space, no significant change in the alveolar-arterial oxygen tension difference both breathing room air and 12 per cent oxygen, no consistent change in oxygen consumption and an increase in the arterial-venous oxygen difference. The calculated cardiac output fell after this procedure, and the pulmonary vascular resistance rose. Although the resection had a detectable effect on the cardiac output and on the pressure in the pulmonary artery, there was no adverse effect on pulmonary ventilation–perfusion relationships, and the diffusing capacity was so high that, even during hypoxia, there was no appreciable difference between the partial pressure of oxygen in the alveoli and in the blood leaving the alveoli.

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A SIMPLE DEFIBRILLATOR ELECTRODE

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THE authors have designed and constructed a simple and convenient defibrillator electrode for use with a modified Mackay defibrillator[1] (Fig. 1). The electrode is made from a stainless steel ladling spoon, a 10 inch length of ⅜ inch OD stainless steel tubing, and a male banana plug. The handle is severed from a spoon, the stub of the spoon handle is silver soldered into one end of the stainless steel tube. A male banana plug is placed into the other end of the rod and silver soldered into place. The back of the spoon is sand blasted and covered with an insulating coat of baked on epoxy resin. The stainless steel rod is insulated by Tygon tubing (formulation B 44 3) of ⅜ inch ID and ⅝ inch OD. This construction permits repeated autoclaving. The

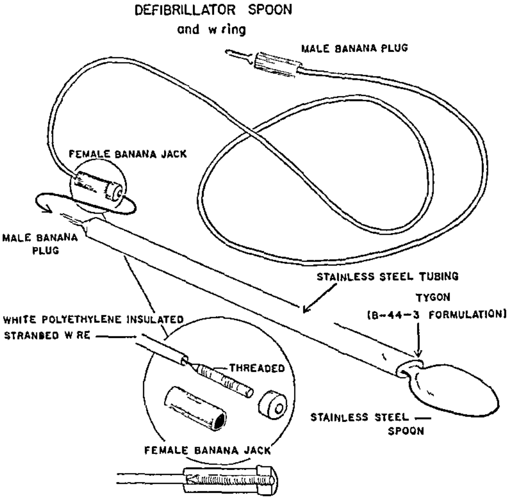

Fig. 1

From Division of Surgery, Army Medical Service Graduate School, WRAMC, Washington, D. C., and Department of Surgery, State University of New York, School of Medicine at N. Y. C., Brooklyn, N. Y.

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electrode is connected to the defibrillator by appropriate wiring using polyethylene-insulated stranded wire, which can be boiled, or Teflon-insulated wire, which can be autoclaved. Banana connectors are used at both ends of the leads. The advantages of this electrode are:

1. It is easily constructed and easy to keep clean. It can be autoclaved.

2. The spoon is properly cupped to make good contact with the heart. It gives sufficient area of contact so that there is minimal danger of excessive current density. The danger of burning the myocardium is thereby minimized.

3. The use of banana jacks allows for rapid setup and use.

4. The back of the spoon is insulated to minimize stray currents.

This design of defibrillator electrodes has been successfully used in experimentation involving use of a mechanical heart.[2]

These electrodes supplied by D. Cole, Glenn Echo, Maryland.

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OCCURRENCE OF TUBERCULOSIS IN AN ACCESSORY PULMONARY SEGMENT

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BOYDEN[1] has described anomalous pulmonary segments Search of the literature has not revealed report of occurrence of tuberculosis in such segments

The occurrence of tuberculosis in an accessory pulmonary segment of Boyden would therefore seem of sufficient interest to report

CASE REPORT

Case No 636 —Twenty year old Negro woman

Diagnosis—Pulmonary tuberculosis, far advanced, chronic, original massive bilateral cavitary disease (Fig 1) with incomplete resolution and assumed inspissation following adequate medical treatment consisting of antituberculosis drugs and bed rest

In the course of the left sided resection the distribution of the tuberculosis disease in the upper lobe was considered unusual The posterior segment was destroyed, as anticipated There was, however, a wedge shaped area of contracted, nodular tissue located between the apical segment and the anterior segment (Fig 2) The posterior segment was uneventfully resected However, on further dissection in the interlobar fissure, a tubular structure was encountered superficial to the pulmonary artery, and was demonstrated to contain air rather than blood It was identified as an anomalous bronchus supplying the triangular area previously described The arterial supply of this area was by a small branch of the anterior segmental artery The venous drainage was by way of intersegmental veins The area was identified as a BX'a subsegment of Boyden (Fig 3), and was uneventfully resected (Fig 4)

The case is presented in view of its practical interest, that is, the importance of alertness on the part of the surgeon with regard to anomalous bronchi especially in the course of segmental resection lest bronchi and arteries be transsected

As Boyden has mentioned, if the operator were to follow the usual method of removing the lingular segments, he would sever bronchus BX'a and its artery, AX'a, thus compromising the whole region posterior to the anterior

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Received for publication Aug 2 1955

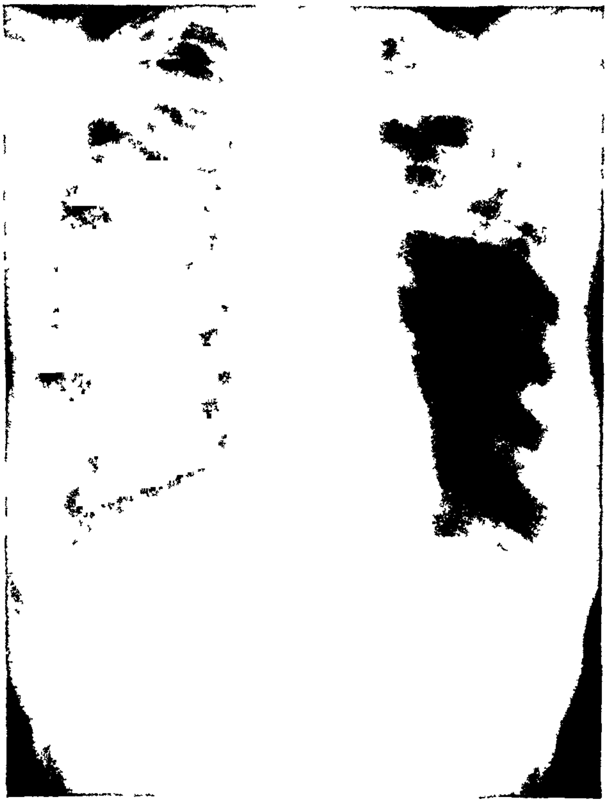

Fig. 1.—Original x-ray

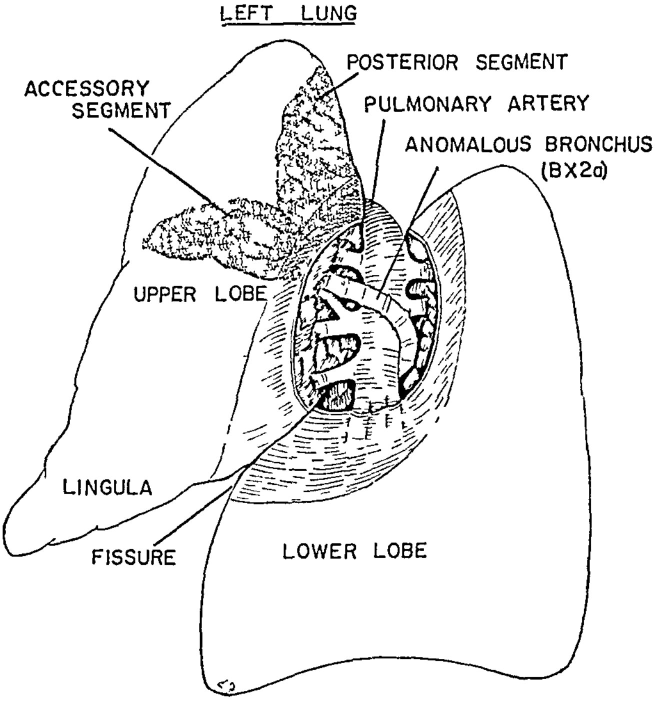

Fig 2.—Diagram showing findings at operation

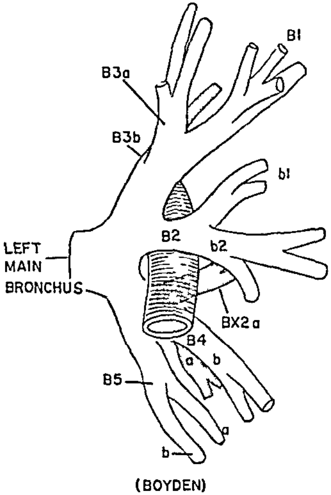

Fig. 3.—Diagram showing accessory bronchus BX2a according to Boyden.

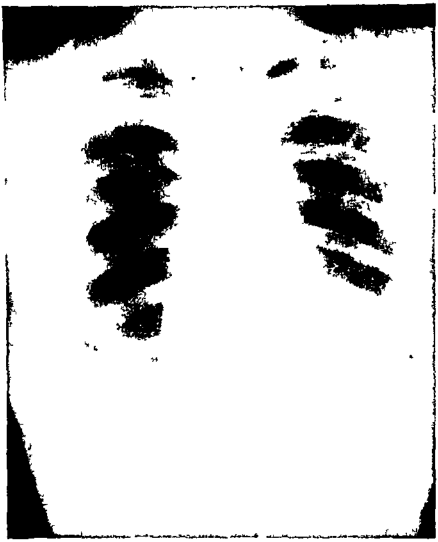

Fig. 4.—Final x-ray following bilateral pulmonary resection.

segment (B^2). Boyden therefore emphasizes the necessity of examining the interlobar surface of the upper lobe before resecting the lingular segments.

The case was of theoretical interest in view of the demonstration of segmental localization of tuberculous involvement, and also in view of the fact that the pulmonary tissues supplied by the aberrant bronchus were involved with infectious disease in spite of the fact that the segments on either side were not significantly involved.

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Original Communications

THE SURGICAL TREATMENT OF LOWER LOBE TUBERCULOSIS

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TUBERCULOUS cavities and bronchial stenosis in a lower lobe are now generally considered indications for primary resection. For these cases, pneumothorax is usually found in my opinion to be a "waste of time and loss of function." The advantage of pneumoperitoneum is that it is mainly only a loss of time. In a great number of cases, it is therefore necessary to perform a primary resection to attain a lasting cure.

After a lower lobe resection, we consider it necessary to diminish the thorax in the following cases:

1. When a segment or more from the middle or upper lobe has to be resected at the same time as the lower lobe lobectomy.
2. If palpable tuberculosis is found in the remaining portion of the lung.
3. If an empyema is resected at the same time.
4. When the tuberculous cavity was opened during the operation.
5. If the remaining lung is very emphysematous or fibrotic.
6. If there is significant contralateral tuberculosis. Following these principles only 2 of 21 patients operated upon for lower lobe tuberculosis were left without a space-diminishing procedure. This paper is concerned mainly with the different surgical possibilities of diminishing the size of the thoracic cavity after a lower lobe resection.

A. *Lower Lobe Resection Without Space-Diminishing Procedure.*—After the removal of a lower lobe with only a localized tuberculous lesion to this lobe,

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This work was aided by The Swedish National Association Against Tuberculosis.

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there is usually no indication for diminishing the size of the thoracic cavity. When both lower and middle lobes are resected and no thoracoplasty performed, the following result was obtained (Case 1, Table I).

TABLE I. LUNG FUNCTION AFTER RIGHT LOWER AND MIDDLE LOBE RESECTION WITHOUT SPACE-DIMINISHING PROCEDURE

CASE NUMBER	RESECTION OF		FUNCTION OF RIGHT SIDE IN % OF TOTAL		YEARS AFTER OPERATION	MOVEMENT OF THE DIAPHRAGM	
	LOBES	RIBS	O_2 UPTAKE	VENTILATION		RIGHT	LEFT
1.—A 22-year-old woman	Right lower and middle lobes	0	26	49	2	1.5 cm.	4 cm.

Conclusion: The remaining overdistended right upper lobe did not show any sign of reactivation after two years and the patient's sputum and gastric washing were negative on culture and guinea pig test. In spite of the overdistention, there was an optimal oxygen uptake of 26 per cent of the total.

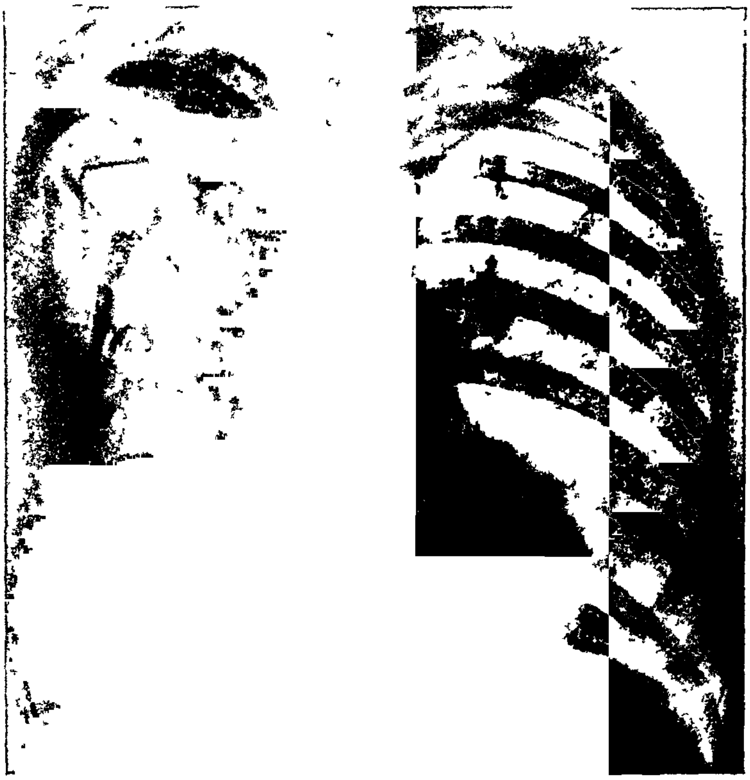

FIG. 1.—Case 2 An x-ray taken three years after resection of right lower and middle lobes with a basal thoracoplasty (resection of ribs 10 to 4). Twenty-four per cent oxygen uptake and 34 per cent of the ventilation come from the remaining right upper lobe.

However, there was an abnormally high ventilation on the right side (49 per cent of the total) in proportion to the oxygen uptake. This is an expression of a very uneconomical respiratory work, due to the overdistention with emphysema.

B. *Diminishing the Space From Below.—*

1. *Basal thoracoplasty:* In 3 cases the thoracic cavity was diminished from below corresponding to the resected lobes, or in other words a basal thoracoplasty was performed. The postoperative function of the remaining lung is shown in Table II (Figs. 1 and 2).

TABLE II. LUNG FUNCTION AFTER BASAL THORACOPLASTY AND LOWER LOBE RESECTION

CASE NUMBER	RESECTION OF		FUNCTION OF RIGHT SIDE IN % OF TOTAL		YEARS AFTER OPERATION	MOVEMENT OF THE DIAPHRAGM	
	LOBES	RIBS	O_2 UPTAKE	VENTILATION		RIGHT	LEFT
2.—A 33 year-old woman	Right lower and middle lobes	10 to 4	24	34	3	1.5 cm.	5 cm.
3.—A 21-year-old woman	Right lower and middle lobes	10 to 7	23	33	2	3 cm.	6 cm.
4.—A 26 year old woman	Right lower lobe	11 to 7	36	43	2	5 cm.	6.5 cm.

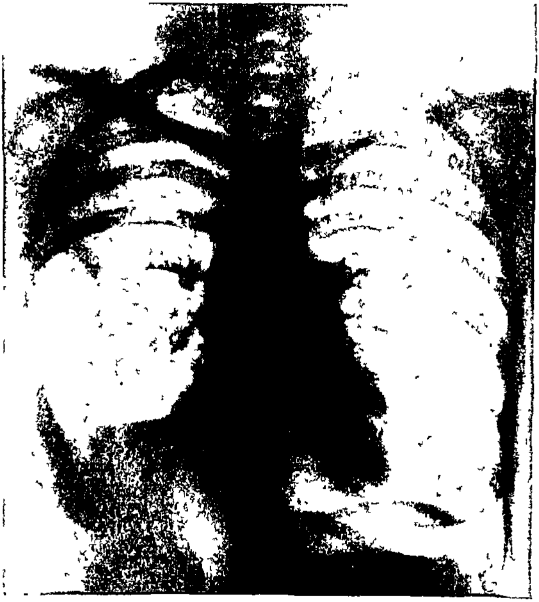

Fig 2.—Case 3. An x-ray taken two years after resection of right lower and middle lobes with a smaller basal thoracoplasty (resection of ribs 10 to 7) which caused considerable deformity. Twenty-three per cent of the oxygen uptake and 33 per cent of the ventilation come from the remaining right upper lobe.

Conclusion With the performance of a basal thoracoplasty after a lower lobe resection all 3 patients had negative cultures and negative guinea pig tests on sputum and gastric washings. An optimal oxygen uptake was obtained by the remaining lung on the operated side at a moderately increased ventilation. Thus, a much more economical respiratory work is obtained as compared with Case 1 in which no space-diminishing procedure was performed. However, a comparatively extensive rib resection had to be performed, resulting in a considerable deformity. After the investigation of these 3 patients, the procedure was abandoned.

2a. *Elevation of the diaphragm by phrenic crush:* To diminish the space after a lower and middle lobe lobectomy by an elevation of the diaphragm seems logical. After such an operation there will be some spontaneous elevation of the diaphragm, especially on the left side. The most simple procedure in order to increase the elevation of the diaphragm is to perform a phrenic crush. This procedure has been used for several years by many surgeons. We are, however, afraid: (1) to impair the patient's ability to raise the sputum in the immediate postoperative period, and (2) to impair lung function by paradoxical movements of the paralyzed diaphragm. Furthermore, emphysema is not prevented as function is regained after six to twelve months.

TABLE III. LUNG FUNCTION AFTER RESECTION OF MEDIASTINAL TUMOR WITH PART OF THE LEFT PHRENIC NERVE

CASE NUMBER	OPERATION	FUNCTION OF LEFT SIDE IN % OF TOTAL		YEARS AFTER OPERATION	MOVEMENT OF THE DIAPHRAGM	
		O_2 UPTAKE	VENTILATION		RIGHT	LEFT
5.—A 15-year-old girl	Extirpation of dermoid with left phrenic nerve	34.1	23.4	5	5 cm.	0 cm.

The loss of function after an irreversible damage to the phrenic nerve is illustrated by Case 5, Table III. Five years after the operation the left diaphragm did not move and only 23.4 per cent of the total ventilation came on the left side. The oxygen uptake, on the other hand, was comparatively much better, i.e., 34.1 per cent of the total in spite of the low ventilation due to the damage of the phrenic nerve. It can be questioned if these values are representative as after the introduction of a Carlens catheter for bronchospirometry, every possibility for pendulum air is excluded, i.e., air, which during expiration, is moving from the right lung over into the left lung. As, however, there was no paradoxical movement of the left diaphragm, it may be assumed that the pendulum air was absent or negligible and therefore the values found at bronchospirometry are considered representative. The drawbacks of an interruption of the phrenic nerve may be avoided by a peripheral mobilization of the diaphragm and a resuturing of the diaphragm with its intact nerve supply at a higher level. I have on several occasions during extrapleural pneumonectomies gone through the diaphragm into the abdomen. The diaphragm has then been resutured at the highest possible level. In one case a pneumonectomy was performed under a 5-rib thoracoplasty. Instead of an extended rib resection, I mobilized the diaphragm and resutured it at the upper border of the sixth rib. The same result would probably have been obtained more easily by a phrenic crush. After a lower and middle lobe lobectomy, however, I think the mobilization and resuturing of the diaphragm is *preferrable to a phrenic interruption.*

2b. *Elevation of the diaphragm by mobilization and resuturing at a higher level:* In the above-mentioned cases of extrapleural pneumonectomies the diaphragm with peritoneum was incised and resutured. This is a more rapid procedure than leaving the peritoneum intact. When an elevation of the diaphragm is performed as a planned operation, however, I leave the peritoneum intact in order to diminish the danger of herniation and infection. First an incision is made as peripheral as possible in the diaphragm (Fig. 3). The separation of the diaphragm from the peritoneum is easily performed in the periphery and can be carried in to the centrum tendineum (Fig. 4). The mobilization of the diaphragm is best started posteriorly, where there is no *danger of opening the peritoneum.* The diaphragm is then resutured, either as in Fig. 5*A*, where it encircled the sixth rib and was sutured to itself and to the intercostal muscles After a subcostal incision under the fifth rib, the diaphragm *may be sutured to the fifth intercostal muscle bundle by a row of* fine interrupted silk stitches as shown in Fig. 5*B*. The latter method (Fig. 5*B*)

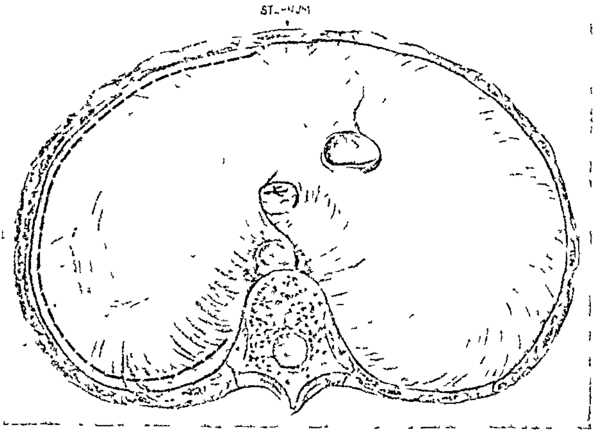

Fig 3.—The diaphragm is incised in the periphery down to the peritoneum.

TABLE IV. LUNG FUNCTION AFTER RESECTION OF RIGHT LOWER AND MIDDLE LOBES WITH ELEVATION OF THE DIAPHRAGM BY MOBILIZATION AND RESUTURING

CASE NUMBER	RESECTION OF	FUNCTION OF RIGHT SIDE IN % OF TOTAL		MONTHS AFTER OPERATION	MOVEMENT OF THE DIAPHRAGM	
		O_2 UPTAKE	VENTILATION		RIGHT	LEFT
6.—A 31 year old woman	Right lower and middle lobes, diaphragm elevation	27.0	32.0	6	0 cm.	5 cm.

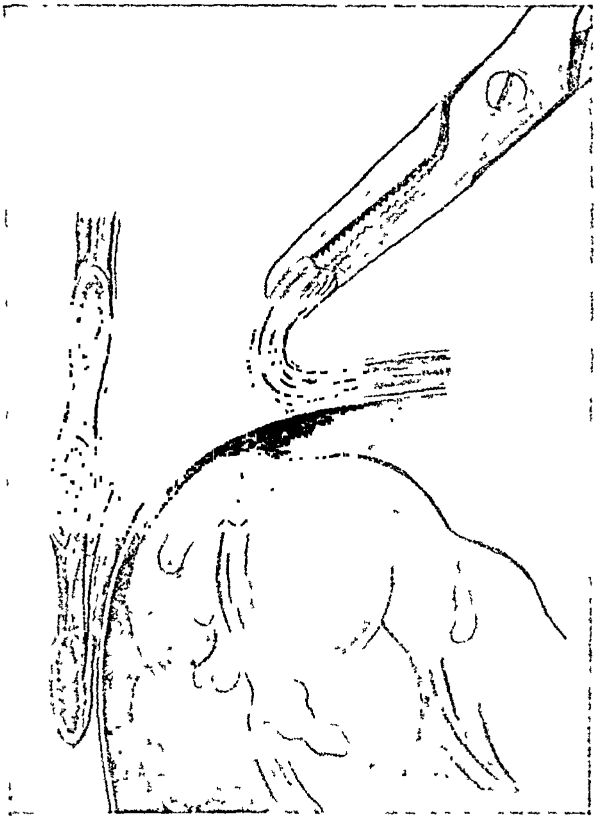

Fig. 4.—The diaphragm is dissected from the peritoneum in to the centrum tendineum.

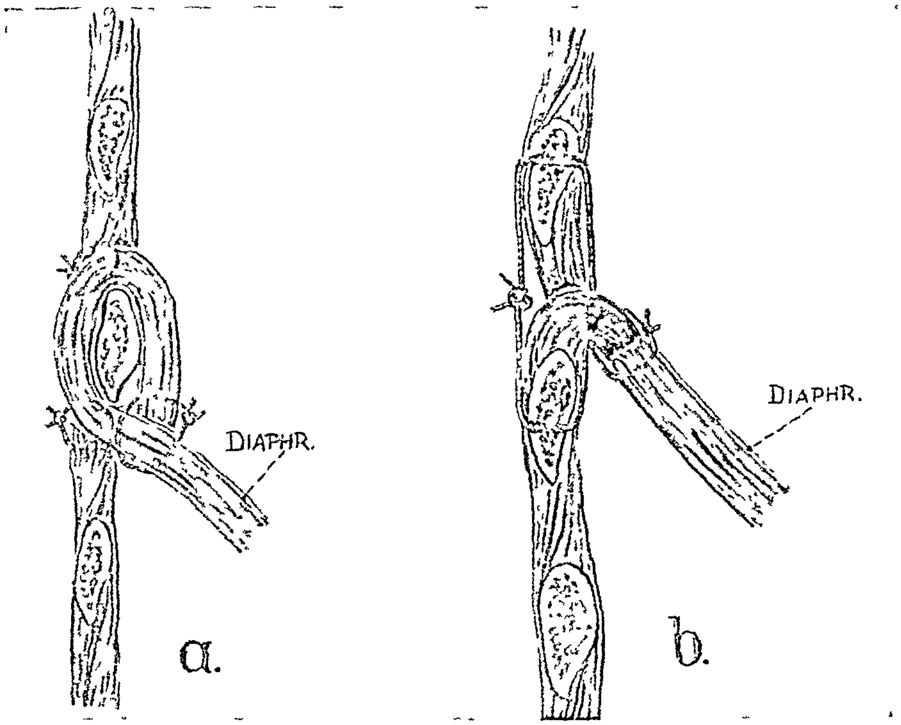

Fig. 5.—The diaphragm is resutured; (a) around a rib, and (b) to an intercostal muscle.

is now preferred. Posteriorly the diaphragm is resutured to the muscles in the intercostal spaces with some heavy nylon sutures being tied around the posterior ends of the ribs (Fig. 6). With intact nerve supply, the resutured diaphragm will act normally in the postoperative period although its excursions are very small. The patients have withstood the procedure well, with an optimum of function and cosmetic result (Table IV, and Figs. 7*A* and 7*B*.) It is difficult, however, to say how much is gained by this more complicated procedure in compari-

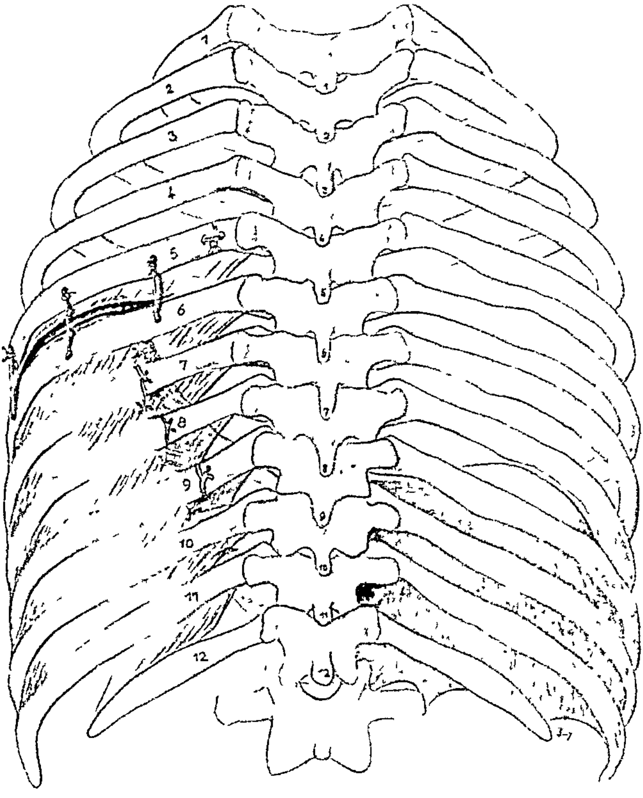

Fig 6.—Posteriorly the diaphragm is resutured to the intercostal muscles and by sutures tied around the ribs

son with a simple interruption of the phrenic nerve. In most cases a simultaneous upper osteoplastic thoracoplasty will be preferred. Only in a few cases where there seems to be no danger of infection may an elevation of the diaphragm be considered.

C. *Diminishing the Space From Above.*—

1. *With an apical extrapleural plombage:* In 4 cases the thorax was diminished by an apical extrapleural plombage. After lobectomy through the bed of the sixth rib, an extrapleural pocket was developed through the bed of the fourth rib and filled with plastic material. There has been no sign of infection for more than one year in these cases. As there is an intact and healthy upper lobe between the extrapleural plombage and the resection area with the divided bronchus the danger of infection should be minimal. The

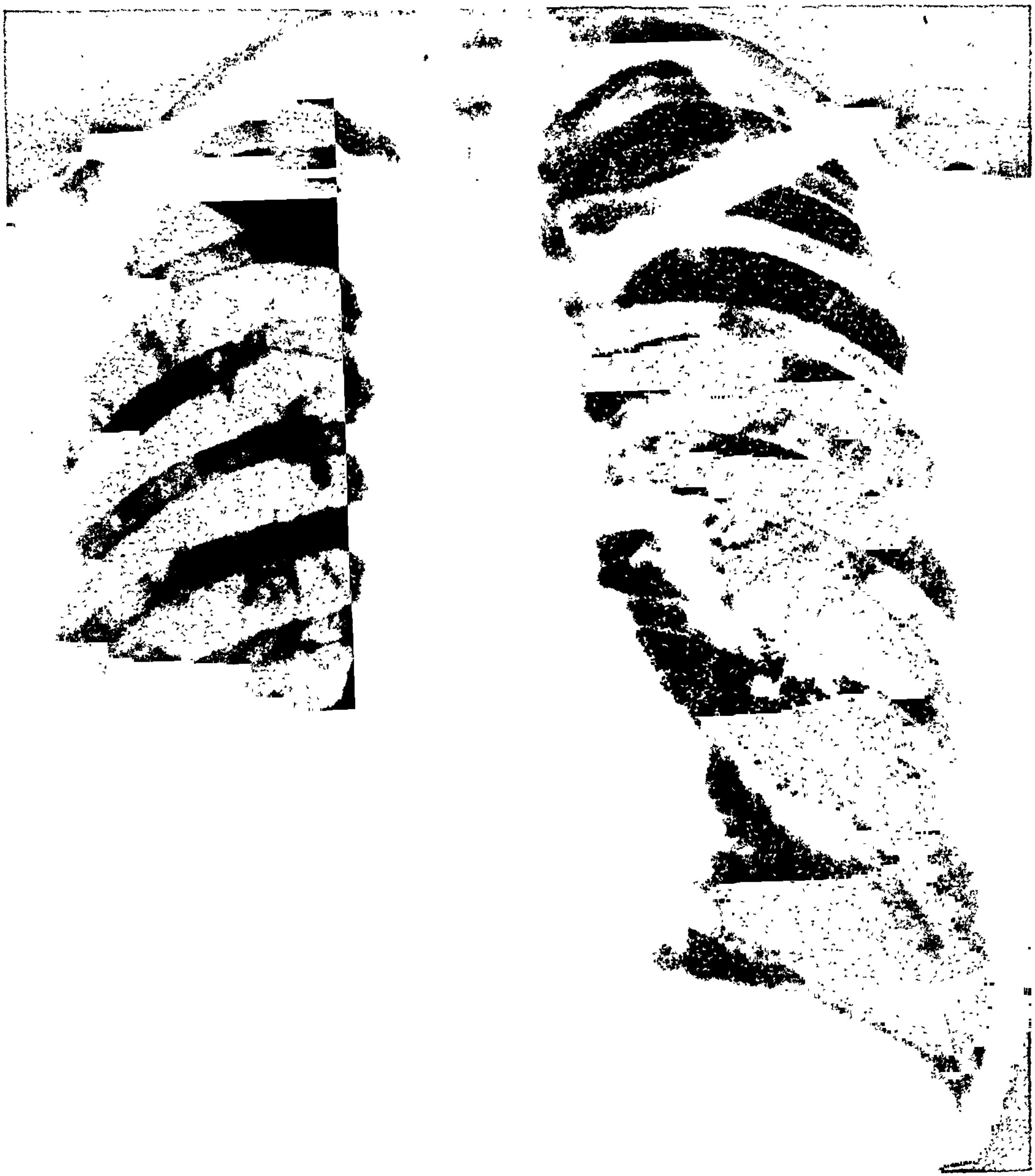

Fig. 7A.

and 7*B*.—Case 6. X-rays taken 6 months after resection of right lower and middle lobes with elevation of the diaphragm after mobilization and resuturing.

very bad experience with plombage on the whole has, however, for the time being made me abandon its use even in these cases. Nor has the functional result obtained encouraged me to further use of plombage (Table V).

TABLE V. THE REMAINING LUNG FUNCTION AFTER APICAL EXTRAPLEURAL PLOMBAGE AND LOWER LOBE RESECTION

CASE NUMBER	RESECTION OF LOBES	FUNCTION OF RIGHT SIDE IN % OF TOTAL		YEARS AFTER OPERATION	MOVEMENT OF THE DIAPHRAGM	
		O_2 UPTAKE	VENTILATION		RIGHT	LEFT
7.—A 20-year-old woman	Right lower and middle lobes	16	29	1		
8.—A 25-year-old woman	Right lower lobe	25	36	1	4 cm.	7 cm.

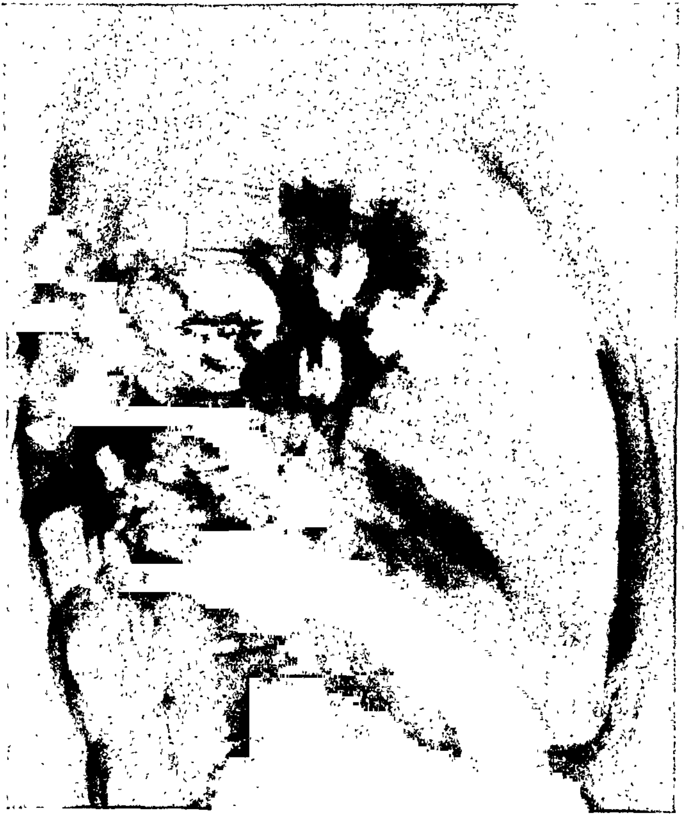

Fig. 7B.—*For legend see opposite page.*

Conclusion: An extrapleural apical plombage in 4 cases has, after one year, not caused any trouble. The patients had negative cultures and negative guinea pig tests with sputum and gastric washings. The oxygen uptake was, however, lower than in the other groups. No definite conclusion can be made about the functional result from a few cases. It is the bad experience with foreign bodies that has made us hesitate to use this procedure.

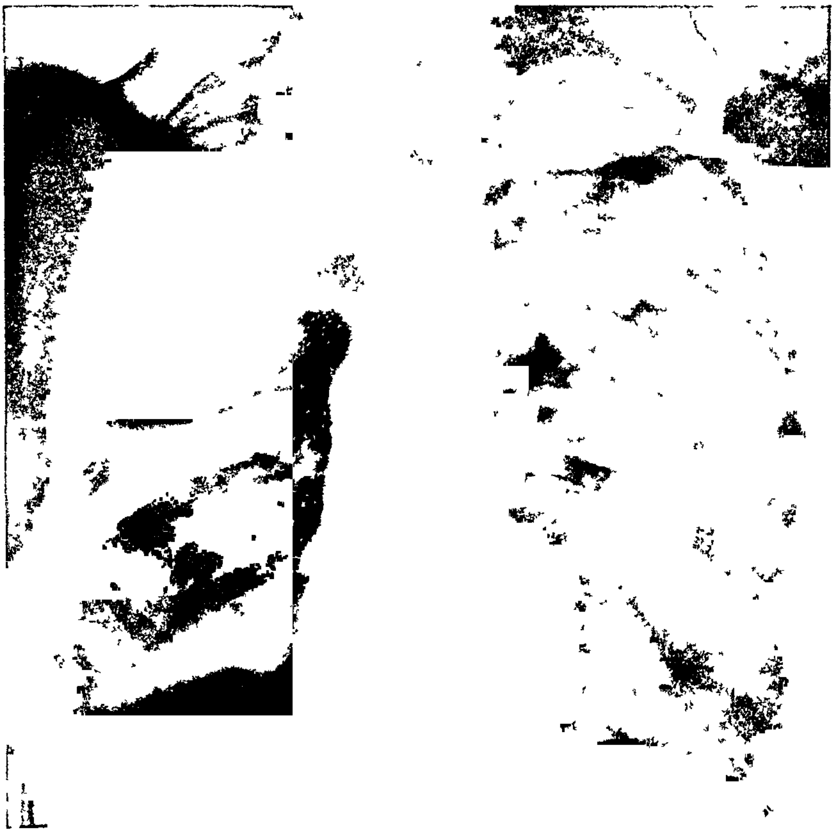

Fig. 8.—Case 7. An x-ray taken one year after resection of right lower and middle lobes with apical extrapleural plombage. Sixteen per cent of the oxygen uptake and 29 per cent of the ventilation come from the remaining right upper lobe.

2. *Primary apical thoracoplasty with lower lobe lobectomy at a second stage:* In order to diminish the space of the thorax, first an upper 5-rib thoracoplasty was performed. At a later date, the diseased lower lobe was resected, usually through the bed of the seventh rib. Five cases were treated according to this principle. The postoperative lung function obtained is shown in Table VI.

Conclusion: A good functional result may be obtained by this two-stage procedure. A one-stage operation is, however, always to be preferred. Furthermore, it must be considered wrong in principle to perform a first stage upper thoracoplasty in a case with lower lobe tuberculosis, as the first operation (a) does not close a cavity in a lower lobe, and (b) will cause a more or less pronounced atelectasis of the healthy upper lobe in the postoperative period with shunting of blood through nonventilated alveoli.

TABLE VI. LUNG FUNCTION AFTER PRIMARY APICAL THORACOPLASTY AND LOWER LOBE RESECTION WITH EXTENDED THORACOPLASTY AT A SECOND STAGE

CASE NUMBER	RESECTION OF		FUNCTION OF RIGHT SIDE IN % OF TOTAL		YEARS AFTER OPERATION	MOVEMENT OF THE DIAPHRAGM	
	LOBES	RIBS	O_2 UPTAKE	VENTILATION		RIGHT	LEFT
9.—A 52-year-old woman	Right lower and middle lobes	Stage I 2, 3 Stage II 4-7	25.3	37.7	1	2.5 cm.	4 cm.

From a physiologic point of view this plan of treatment must be considered unsatisfactory and has been abandoned by me.

3. *One-stage lower lobe resection and upper osteoplastic thoracoplasty:* In the last 10 cases of lower lobe tuberculosis in which it has been found desirable to diminish the thorax, an apical osteoplastic thoracoplasty has been performed at the same time as the lung resection.

This procedure is facilitated by operating on the patient in the face-down position with the upper part of the body tilted downward. In this way a subcostal incision under the fifth rib may be used. In this position, the lower lobe is pushed down into the incision by the elevated diaphragm. If the lower pulmonary vein is first divided, the dissection of the lower lobe bronchus and artery is facilitated and there is no danger of damage to the middle lobe structures. When it is decided to complete the operation with a thoracoplasty, the posterior ends of ribs 2 to 4 are also divided to give a better exposure. After completion of the resection, the thoracoplasty is performed in the following way: Posterior parts of ribs 2 to 5 are resected in increasing lengths from above and downward (Fig. 9). Rib 2 is bent in the costal cartilage and fixed with a nylon suture through a drill hole and through the costovertebral ligament and behind the head of the rib on the fifth or sixth vertebra (Fig. 10).

The second rib should, when fixed, be as close to the mediastinum as possible. To obtain a fixation close to the mediastinum it is necessary to make an incision in the cartilage of the second rib near the sternum (Fig. 11). Then a row of 5 to 8 isolated sutures are used to fix the first intercostal muscle bundle to the mediastinal pleura in order to prevent any re-expansion of the lung medial to the new roof. The posterior ends of ribs 3 to 5 are fixed by nylon sutures through drill holes to the posterior end of the sixth rib. One nylon suture anteriorly through drill holes in the fifth and sixth ribs will approximate these ribs and then usually no more sutures are needed for the closure of the chest wall in this plane. It is not necessary to perform an apicolysis. The pleura may be incised at the lateral border of the first rib which is left intact. The mediastinal pleura is then left in place above the new roof. When intact, the mediastinal pleura may be carried down to cover the bronchial stump, the resected area of the lung, and to separate the space above and below the roof. For drainage, two tubes are left intrapleurally and one is left above the new roof. The functional result is shown in Table VII.

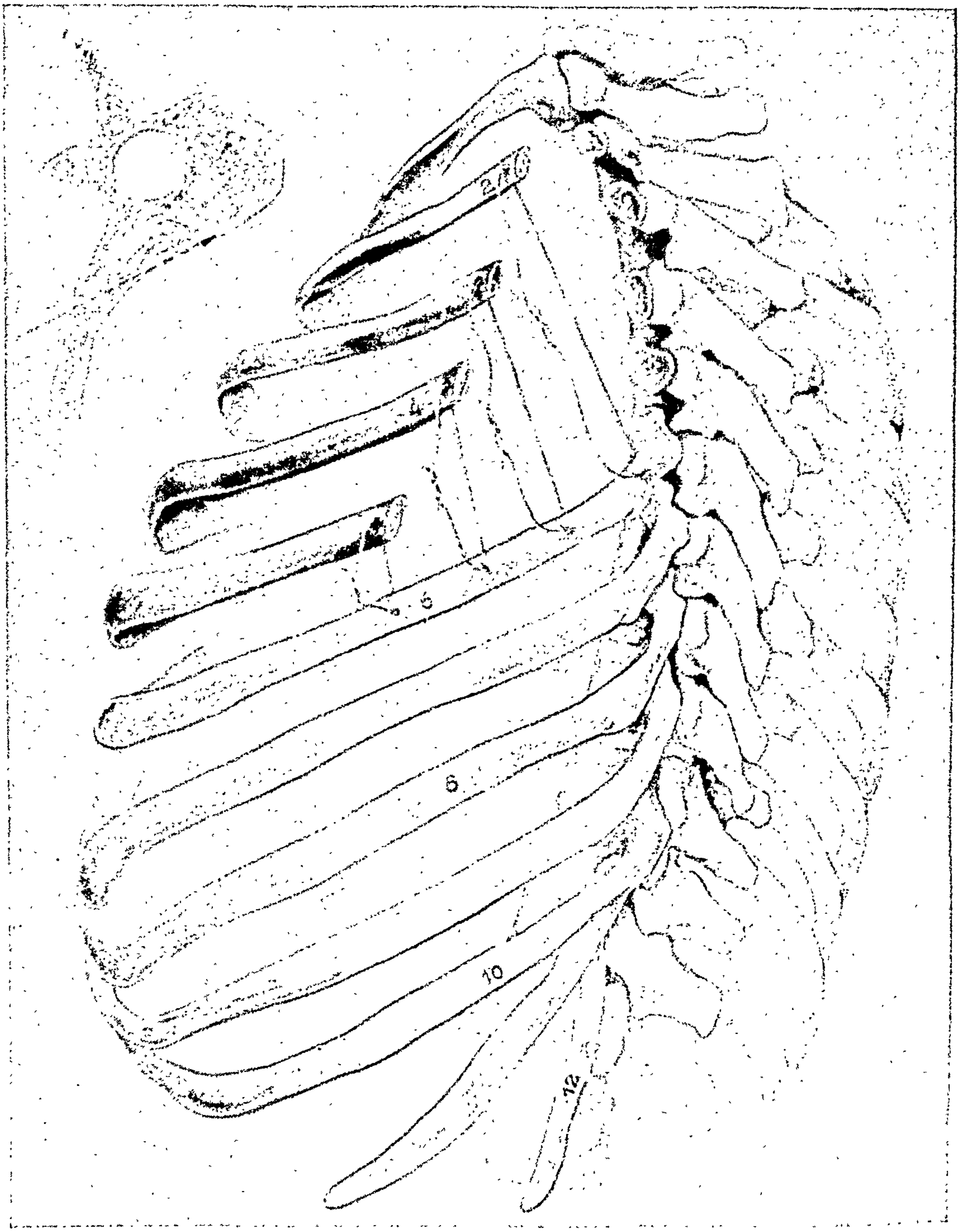

Fig. 9.—Posterior parts of ribs 2 to 5 are resected in increasing lengths from above and downward.

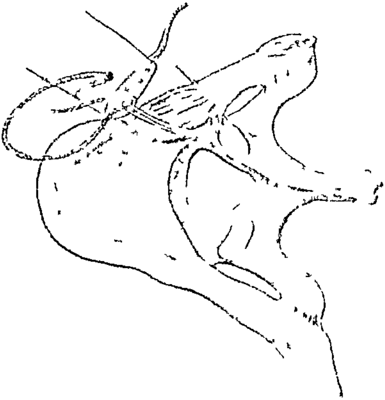

Fig 10 —A diagram showing the fixation of the second rib with a nylon suture through a drill hole and passed behind the head of the fifth or sixth rib

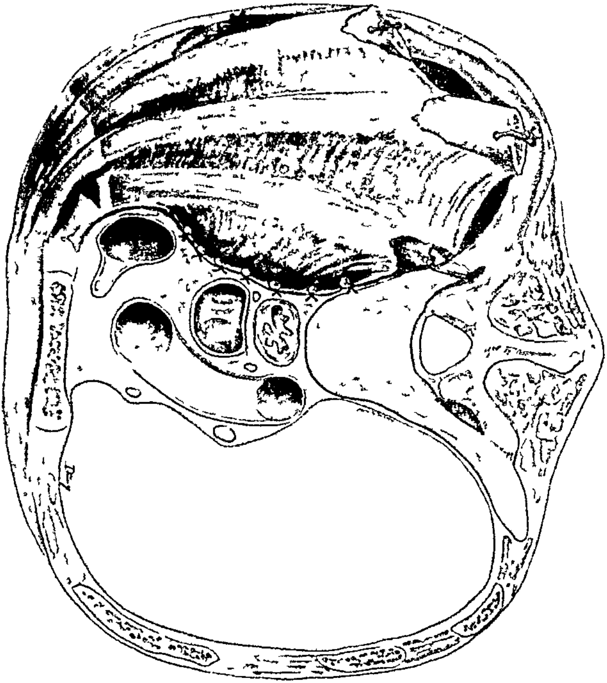

Fig 11 —The new roof seen from above The ribs are bent in the costal cartilages and fixed posteriorly by nylon sutures through drill holes A row of isolated sutures fixes the first intercostal muscle bundle to the mediastinal pleura. Note the incision anteriorly in cartilage of the second rib

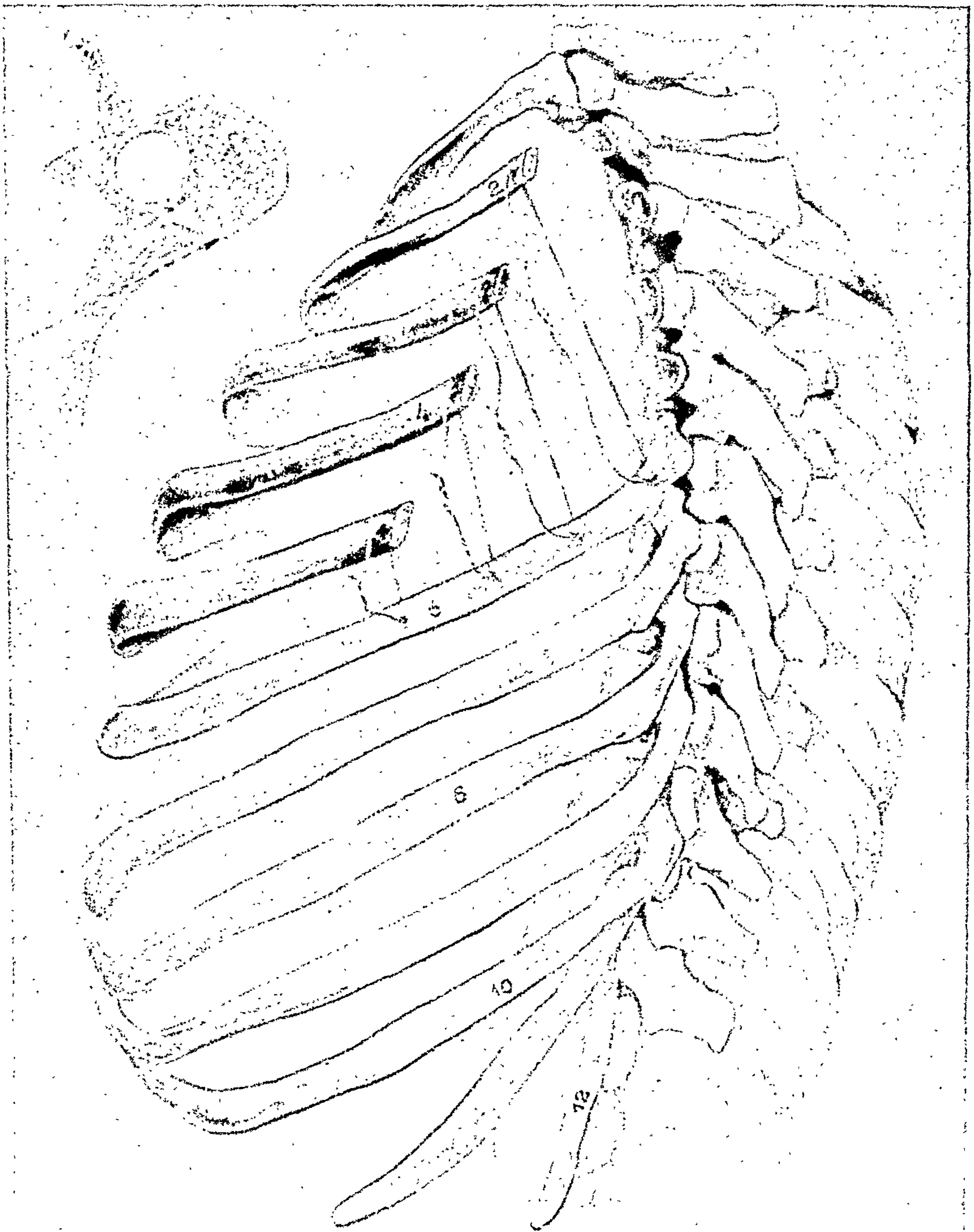

Fig. 9.—Posterior parts of ribs 2 to 5 are resected in increasing lengths from above and downward.

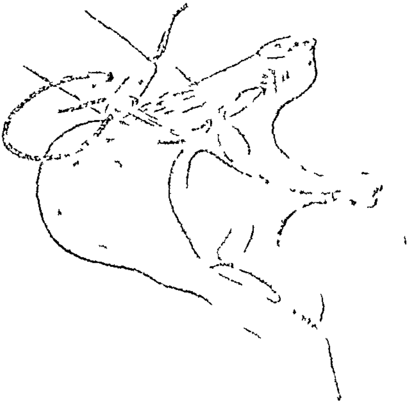

Fig 10 — A diagram showing the fixation of the second rib with a nylon suture through a drill hole and passed behind the head of the fifth or sixth rib

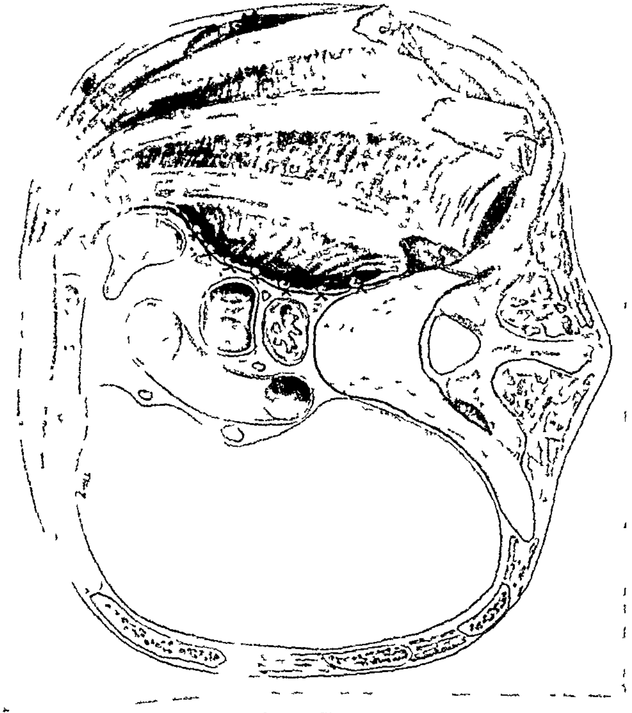

Fig 11 The new roof seen from above The ribs are bent in the costal cartilages and fixed posteriorly by nylon sutures through drill holes A row of isolated sutures fixes the first intercostal muscle bundle to the mediastinal pleura. Note the incision anteriorly in cartilage of the second rib

TABLE VII. LUNG FUNCTION AFTER ONE-STAGE LOWER LOBE RESECTION AND UPPER OSTEOPLASTIC THORACOPLASTY

CASE NUMBER	RESECTION OF		FUNCTION OF RIGHT SIDE IN % OF TOTAL		YEARS AFTER OPERATION	MOVEMENT OF THE DIAPHRAGM	
	LOBES	RIBS	O_2 UPTAKE	VENTILATION		RIGHT	LEFT
10.—A 30-year-old woman	Right lower and middle lobes	2-6 fixed on 7th	24.1	34.2	1	5 cm.	6 cm.
11.—A 22-year-old man	Right lower and middle lobes	2-6 fixed on 7th	25.1	38.0	1	4 cm.	6 cm.
12.—A 19-year-old man	Left lower lobe	2-6 fixed on 7th	77.0	77.0	1	8 cm.	5 cm.

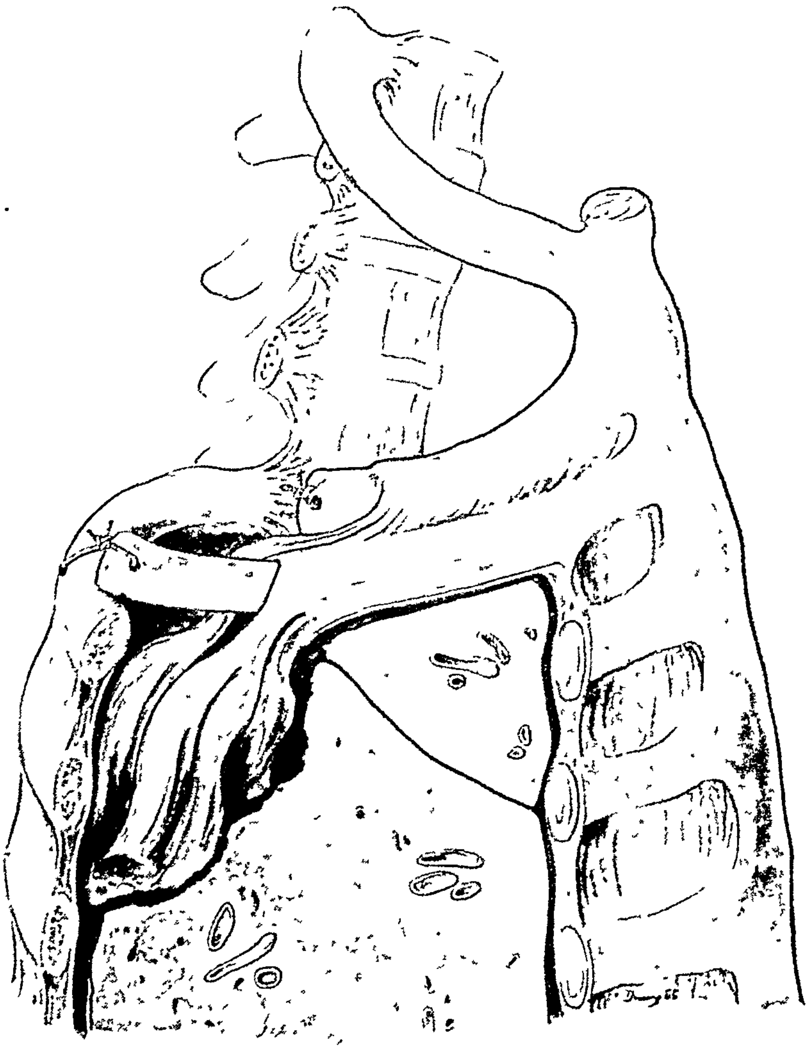

Fig. 12.—A diagram showing the flap of intercostal muscle bundles, which in case of segmental resection is placed directly on the resected area. This muscle flap will diminish the incidence of air leakage from the resected area and make the postoperative treatment easier.

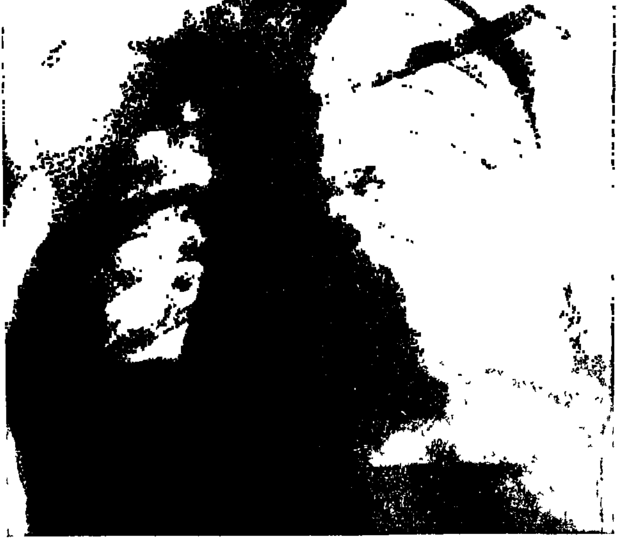

Fig. 13.—Case 10 An x-ray taken one year after resection of right lower and middle lobes with osteoplastic thoracoplasty Twenty-four per cent of the oxygen uptake and 34 per cent of the ventilation come from the remaining right upper lobe

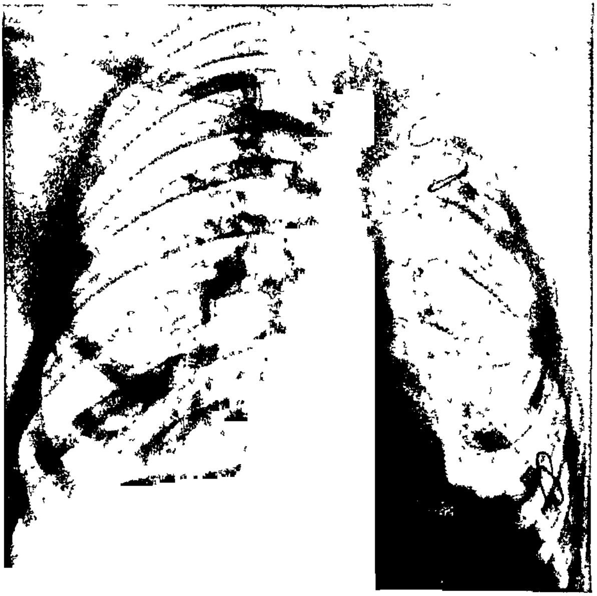

Fig 14 —Case 12. An x-ray taken one year after resc with
osteoplastic thoracoplasty Twenty-three per cent of the oxy of
the ventilation come from the remaining left upper

TABLE VII. LUNG FUNCTION AFTER ONE-STAGE LOWER LOBE RESECTION AND UPPER OSTEOPLASTIC THORACOPLASTY

CASE NUMBER	RESECTION OF		FUNCTION OF RIGHT SIDE IN % OF TOTAL		YEARS AFTER OPERATION	MOVEMENT OF THE DIAPHRAGM	
	LOBES	RIBS	O_2 UPTAKE	VENTILATION		RIGHT	LEFT
10.—A 30-year-old woman	Right lower and middle lobes	2-6 fixed on 7th	24.1	34.2	1	5 cm.	6 cm.
11.—A 22-year-old man	Right lower and middle lobes	2-6 fixed on 7th	25.1	38.0	1	4 cm.	6 cm.
12.—A 19-year-old man	Left lower lobe	2-6 fixed on 7th	77.0	77.0	1	8 cm.	5 cm.

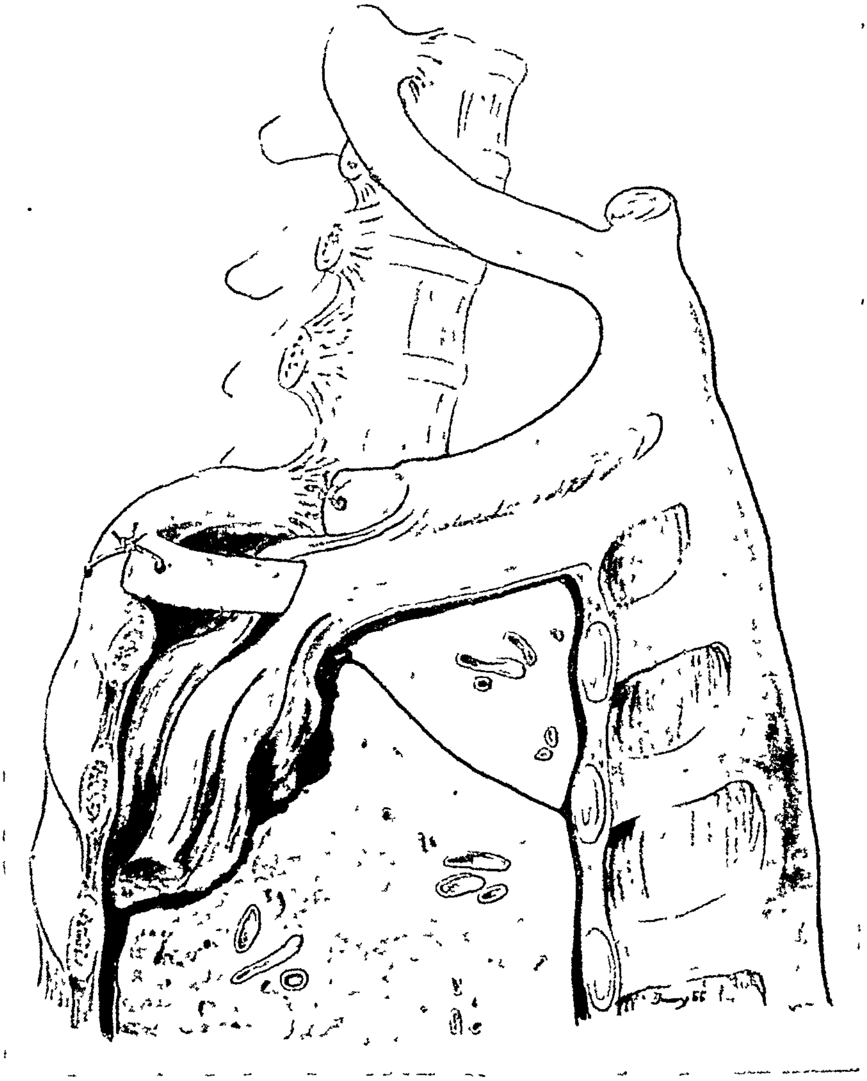

Fig. 12.—A diagram showing the flap of intercostal muscle bundles, which in case of segmental resection is placed directly on the resected area. This muscle flap will diminish the incidence of air leakage from the resected area and make the postoperative treatment easier.

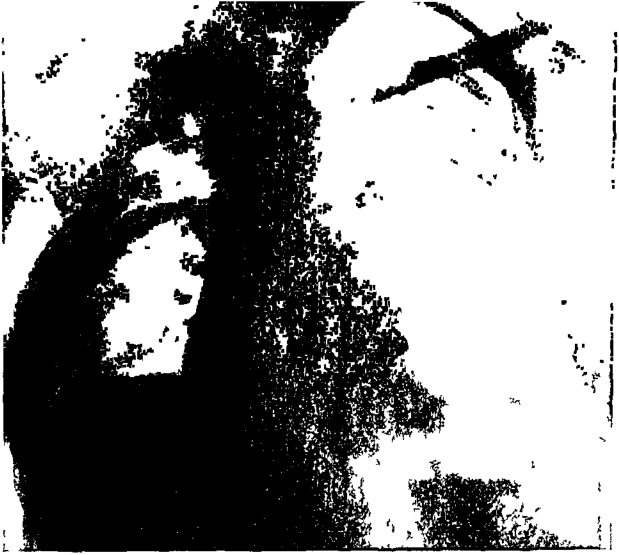

Fig 13 —Case 10 An x ray taken one year after resection of right lower and middle lobes with osteoplastic thoracoplasty Twenty four per cent of the oxygen uptake and 34 per cent of the ventilation come from the remaining right upper lobe

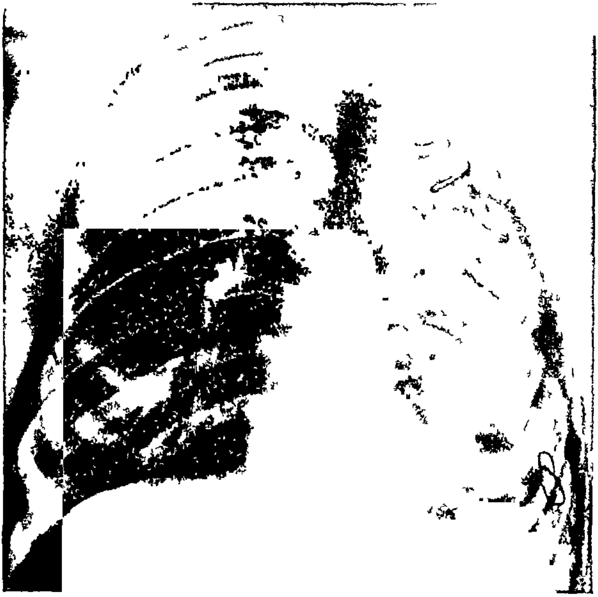

Fig 14 —Case 12 An x ray taken one year after resection of left lower lobe with osteoplastic thoracoplasty Twenty three per cent of the oxygen uptake and 23 per cent of the ventilation come from the remaining left upper lobe

Conclusion: In all these one-stage procedures of lower lobe resection with upper osteoplastic thoracoplasty, I used in the beginning the lateral position making the incision through the bed of the sixth rib and making the roof on the seventh rib. In the face-down position, however, a lower lobectomy is easily carried out through the bed of the fifth rib permitting a more horizontal roof with fixation of the ribs to the posterior end of the sixth rib. From a functional point of view the results obtained were as good by this one-stage procedure as in the earlier two-stage procedures. Furthermore, the same function was obtained by this upper osteoplastic thoracoplasty as by the earlier basal thoracoplasties. The cosmetic result after an upper osteoplastic thoracoplasty is, however, far superior, as the deformity is mainly localized to the axilla, and cannot be seen unless the undressed patient lifts the arm.

DISCUSSION

In cases of lower lobe resection where the remaining lung parenchyma is healthy, the chest may be left intact. The resulting overexpansion will increase the minute ventilation and thus the respiratory work for the same oxygen uptake on the side on which the operation had been performed.

A primary basal thoracoplasty will give a good functional result. The severe deformity following such a procedure has, however, made me abandon its use.

Diminishing the thoracic cavity from above, thus displacing the upper lobe downward to the diaphragm, will usually give a good functional result.

A comparatively poor functional result was obtained when an apical extrapleural plombage was used. A plastic plombage placed above a healthy lobe has so far not caused any trouble. The method is, however, at present abandoned in favor of the one-stage lower lobe resection with osteoplastic thoracoplasty, as the latter method seems to give a better functional result. An elevation of the diaphragm will, however, give as good or even better result, but has so far only been used in a few clean cases. (Only the functional values of cases with unilateral disease are given, in which, from a functional point of view, insignificant nodules were found in the remaining lung on the side on which the operation had been performed.)

SUMMARY

In order to diminish the thoracic cavity in cases where this was indicated after a lower lobe resection, a preliminary basal thoracoplasty was carried out. The resulting deformity has prevented further use of this method. Then the remaining upper lobe has been displaced downward by an apical extrapleural plombage and a primary upper thoracoplasty. The method of choice, however, seems to be an upper osteoplastic thoracoplasty performed at the same operation as the lower lobectomy. The face-down position will facilitate resection of the lower lobe through the bed of the fifth rib, making the roof on the sixth rib. Both the cosmetic and functional results seem to be very good by this one-stage procedure. Only in cases where there is no danger of infection has the diaphragm been mobilized and resutured at a higher level. Then an optimal cosmetic and functional result may be obtained.

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BILATERAL RESECTION IN PULMONARY TUBERCULOSIS

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HITHERTO, patients who became candidates for lobectomy in the treatment for pulmonary tuberculosis were mainly those having a tuberculous focus on one lung only. When pathologic changes existed bilaterally, other methods were used with collapse therapy of the lung.[2, 3] Recently, as lobectomy has become a safe procedure, and as the increased knowledge of pulmonary function has gradually added to the treatment of this disease, unilateral resection with contralateral collapse therapy or bilateral resection has gradually come into use for the treatment of pulmonary tuberculosis. In a rural sanatorium of Japan where I am located, I see quite a number of patients in the outpatient clinic where such treatment is indicated.

Since Eloesser's report[4] in 1932 on bilateral resection, there have been many reports on the treatment of bronchiectasis (Overholt,[5] O'Brien,[6] Kergin,[7] Bisgard,[8] and others). Overholt used this method for the treatment of pulmonary tuberculosis, and since then Chamberlain,[10] Lowell and Conklin,[11] and Shumway and his co-workers[12] have reported nearly a score of such cases.

In Japan, Shinoi[13] and I made separate reports on this problem at the Sixth Annual Meeting of the Japanese Thoracic Surgical Association. In my first case, the patient had large cavities in both of the lower lobes, and artificial pneumoperitoneum was performed for a year without results. Chemical therapy was tried also for several months, without adequate result. Therefore, a bilateral lobectomy on both lower lobes was performed in 1951. Since then I have performed such an operation on 28 patients at the National Okayama Sanatorium. Twenty-two patients who were operated upon by October, 1954, will be reported upon here, and from these results the value of this operation, as well as the preliminary examinations, will be discussed.

SELECTION OF PATIENTS

In performing bilateral resection, the dimension of the lesion, its site, the thickness of the pleural membrane, the movement of the diaphragm, the condition of the bronchi, and the patient's general condition should be considered. A separate bronchospirometric examination for each lung is needed before the operation.

Preoperative Chest X-ray Findings (Figs. 1-5).—In all of the cases reviewed here, the dimension of the lesion was limited to one lobe and the character

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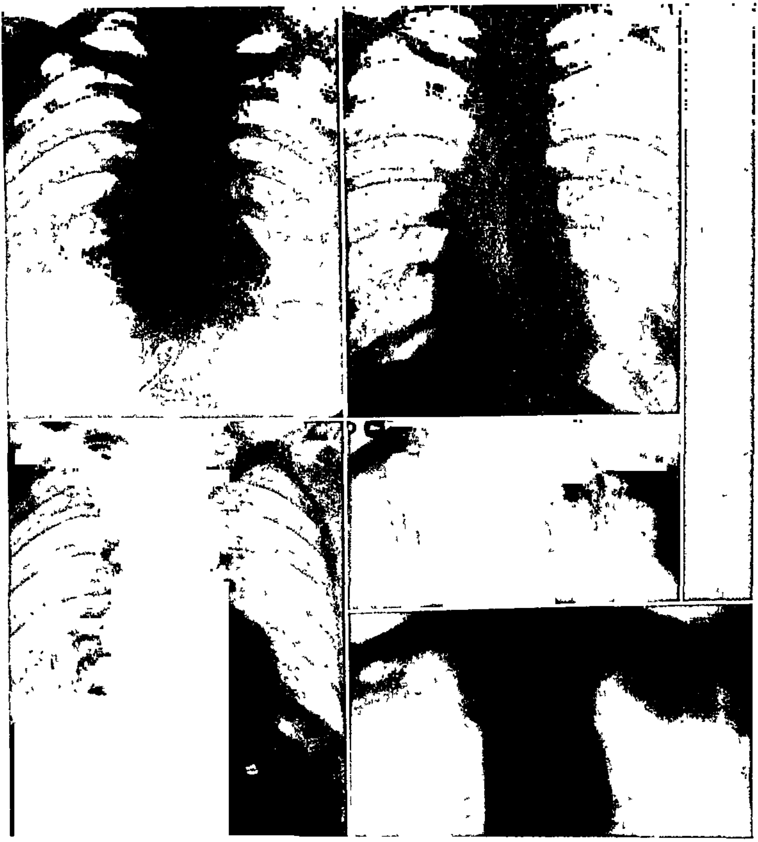
of the lesions were: (a) cavities, (b) localized caseous masses, so-called tuberculomas, and (c) tuberculous bronchiectasis. When they were divided according to the type of case, 7 patients had bilateral cavities (of which one was tuberculous bronchiectasis), 9 had a cavity on one side and localized tuberculous changes on the other, 5 had localized tuberculous changes on both sides, and 1 had an extensive thickening of the pleural membrane. According to the classification of the National Tuberculous Association, 4 cases were far advanced, 12 were moderately advanced, and 6 were minimal (Table I).

The patients were from 18 to 40 years of age. When they were divided according to sex, 15 were males and 7 were females. The time from the onset of disease to the first operation was 6 months to 6 years, 9 months, an average



Figs. 1-5.—Preoperative x-ray find

another both upper lobes and the left S6 were resected (8 segments and 44 per cent of the lung removed). After 2 years and 10 months, respectively, these patients are still working without difficulty.

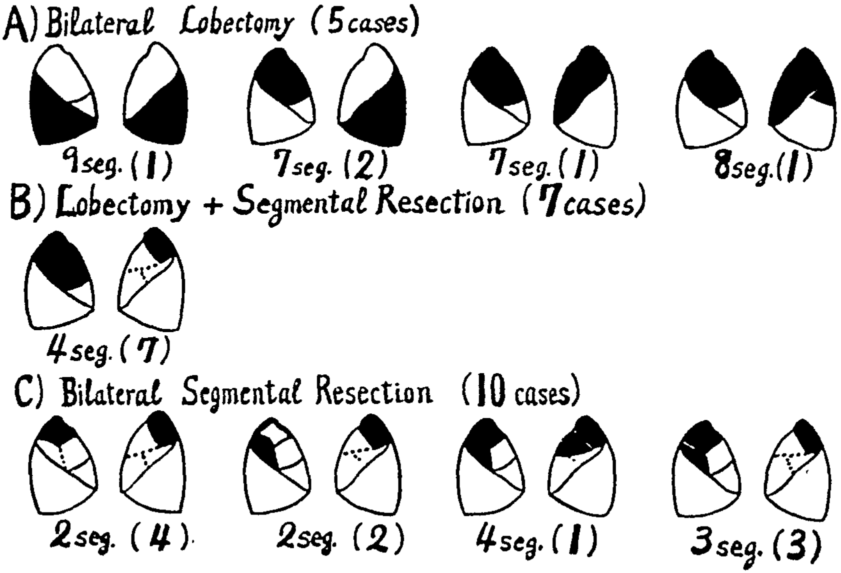

A) Bilateral Lobectomy (5 cases)
9seg. (1)
7seg. (2)
7seg. (1)
8seg. (1)
B) Lobectomy + Segmental Resection (7 cases)
4seg. (7)
C) Bilateral Segmental Resection (10 cases)
2seg. (4)
2seg. (2)
4seg. (1)
3seg. (3)

Fig. 8.—Extent of resections.

THE OPERATION

1. *The Method of Operation.*—Due to the problem of preserving pulmonary function and to the advances in chemotherapy, there have been many opinions as to whether thoracoplasty should be performed after segmental resection or lobectomy. In my cases where lobectomy of both upper lobes was performed, obliterative thoracoplasty was not done. When, however, a large cavity existed or the patient had a tuberculous bronchiectasis on one side, a planned thoracoplasty was performed a month before the main resection in order to prevent any postoperative complications.[15, 16] I have found from experience that when there was a tuberculous focus in the left upper lobe, resection of S_{1+2} and S_3 was perferable in many cases to a complete lobectomy.

2. *The Elapse of Time Between the Two Operations.*—There was a period between operations of cases, 3 to 6 months in 7 cases, and under 3 months in 6 cases. When a bilateral resection was planned and if the patient's condition after the first operation was good, it was usually possible to perform the second lobectomy about 4 months after the first one; in cases of segmental resection it was 3 months. Simultaneous bilateral segmental resection, started by Overholt, is possible if the patients are picked carefully according to other factors (Shumway and associates[12]). I had 2 cases where simultaneous bilateral segmental resection (S_1, and S_{1+2}) was performed and the postoperative course was uneventful, without either dyspnea or cyanosis. Both of the patients had fully recovered at the time of their discharge from the hospital. The selection of patients should be done very carefully and the condition of the patient after the first operation should be considered before applying this method. It is best not to prepare for a simultaneous bilateral resection at the beginning.

3. *Picking the Site of the First Operation.*—It is generally believed that the side which has the larger focus should be operated upon first. By removal

of the major focus of the disease, a large contagious focus may be removed which will help the patient to recover function. Furthermore, this would decrease the operative wound on the contralateral side of the lung. When the degree of the disease between both sides is about the same, the anatomic relation and the influence of the cardiac function should be considered and usually the right side is the one operated upon first.

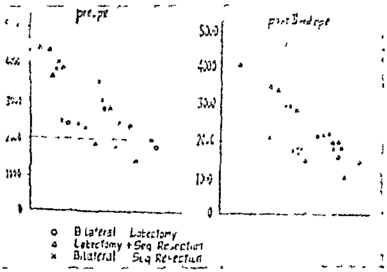


Fig. 9.—Pre- and postoperative vital capacity in bilateral resection.

THE POSTOPERATIVE PULMONARY FUNCTION

As O'Brien,[6] Mannix,[17] and Hayashi[19] have already stated, of all the causes of postoperative disturbances in the pulmonary function, the main danger is from pleural complications. In these, the thorax and its contents, the lung parenchyma, bronchi, and the chest walls are all involved. The postoperative re-expansion of the lung is a more serious problem in bilateral pulmonary resection than in a unilateral resection. The surgeon must also know how much pulmonary function the individual must recover to be able to return to his ordinary life. The operation is only possible when an adequate pulmonary function is assured after the operation. Kano,[3] as a result of his experience in bilateral thoracoplasty, states that a vital capacity of 1,500 c.c. is needed for the individual to lead an ordinary life.

Fig. 9 shows the vital capacity changes before and after operation. There is only 1 case (a woman) where the vital capacity was below 1,500 c.c. after a bilateral pulmonary resection. Most of the patients who had bilateral segmental resection (Group C), and unilateral lobectomy with a contralateral segmental resection (Group B) had a vital capacity of over 2,000 c.c. Their volume loss in vital capacity was smaller than those who had had a bilateral lobectomy (Group A). The volume loss in the vital capacity to that of the preoperative value and the rate of decrease are shown in Fig. 10. In Group A, as the vital capacity was smaller than that of Group B, actually the volume of loss was also small, but the rate of decrease paralleled the amount of resection. Those in Group C, where collapse therapy was performed before the operation (Group $C_{(b)}$) there was rather an increase in the vital capacity after the operation.

When the vital capacity and the maximum breathing capacity were obtained with spirometry, the average value in 12 cases was, respectively, [illegible] c.c. and 60 L. per minute, and the percentage to the p[illegible]rmal val lower than 70. This was especially low in Group A (Fig. 11). But, this result was higher than those receiving bilateral extrapleural pneumothorax (VC 60 per cent, MBC 50 per cent), or bilateral thoracoplasty (average of 3 cases, VC 30 per cent, MBC 40 per cent), and from the point of pulmonary function it was far better.

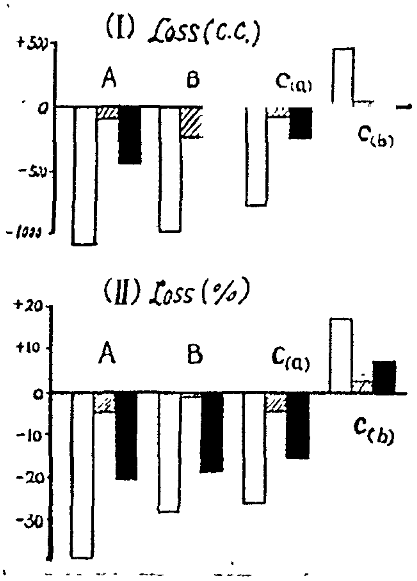


Fig. 10.—Loss of vital capacity with bilateral resection. Maximum ☐ ; Minimum ▨ ; Average ■ .

BRONCHOSPIROMETRY BEFORE AND AFTER OPERATION

Iwabuchi examined the pulmonary function using a Carlens catheter before and after operation in various kinds of chest operations, and his results for bilateral pulmonary resection are shown in Table II.

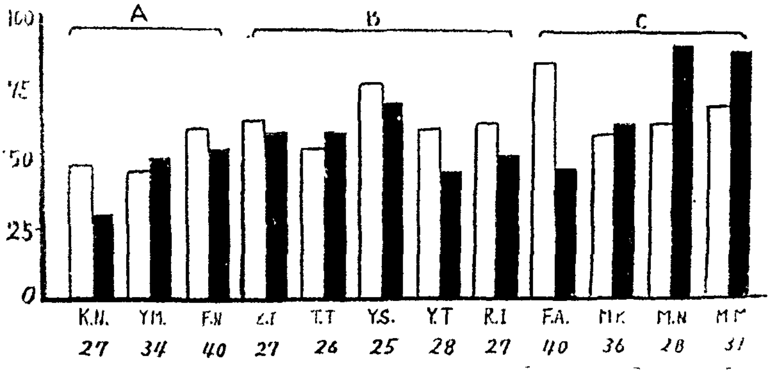


Fig. 11.—Postoperative ventilatory function; MBC and VC, as per cent of predicted (VC average, 2,326 c.c.; MBC average, 60 L. per min.). VC ☐ ; MBC ■ .

There was a decrease in oxygen consumption in all of the groups but there was no significant difference between the three. The decrease in the minute ventilation was especially large in Group A, but in Groups B and C they did not differ much from the value obtained before the operation, showing just a slight decrease. The respiratory count slightly decreased in Group B, but

TABLE II BRONCHOSPIROMETRY IN PRE AND POSTOPERATIVE BILATERAL RESECTION

		(A) BILATERAL LOBECTOMY		(B) UNILATERAL LOBECTOMY CONTRALATERAL SEG. RESECTION		(C) BILATERAL SEGMENTAL RESECTION	
		RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT
O Consumption (c.c.)	Pre	202 (54%)	171 (46%)	174 (53%)	153 (47%)	181 (61%)	119 (39%)
	Post	178 (61%)	114 (39%)	162 (49%)	171 (51%)	171 (58%)	126 (42%)
Minute ventilation (L.)	Pre	6.3	4.4	6.6	5.9	6.3	3.9
	Post	6.3	5.3	6.7	5.8	6.2	3.9
Respiratory frequency	Pre	18	18	19	19	17	17
	Post	19	19	23	23	17	17
Ventilation equivalent	Pre	4.1	4.3	3.8	3.8	3.5	3.3
	Post	3.5	4.6	4.3	3.6	3.7	3.1
Vital capacity (c.c.)	Pre	1,847 (52%)	1,473 (48%)	1,783 (52%)	1,677 (48%)	1,405 (63%)	820 (37%)
	Post	1,417 (55%)	1,143 (45%)	1,250 (50%)	1,277 (50%)	990 (63%)	615 (37%)

Groups A and C were the same as before the operation. The value of ventilation equivalent was about the same as before the operation, and vital capacity paralleled pulmonary resection.

In Figs. 12 and 13 are shown the comparison of vital capacity (tidal volume, expiratory reserve volume, inspiratory reserve volume) and oxygen consumption between the three groups with the results from a typical case representing each group. The ability for each individual to live a normal life after the operation should be judged by his actual mode of life, but as this is quite difficult to do, a load-adding exercise performed after the operation could be used as a gauge. Spirometry before and after exercise is shown in Table III.

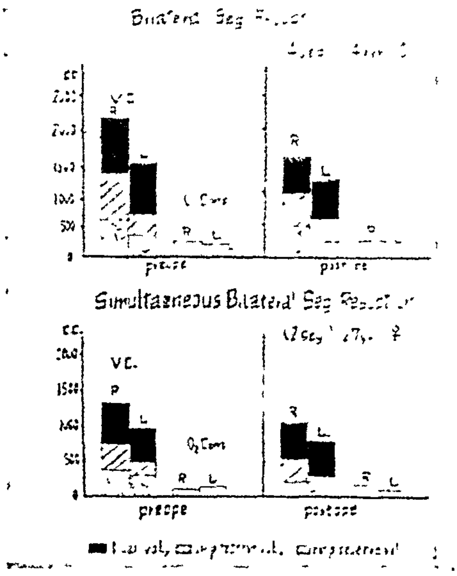


Fig. 12.

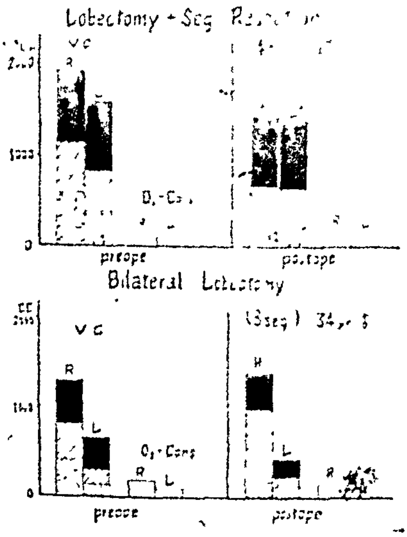


Fig. 13.

Figs. 12 and 13.—Pre- and postoperative bronchospirometry.

and both the oxygen consumption and the minute ventilation were increased in all of the three groups. And in Groups B and C, the value was similar to that of before the operation. The increase in the respiratory rate and pulse rate were below 4 per minute and there was only a slight increase in the ventilation equivalent. MBC increased by 9 L. per minute in Groups A and B, and in Group C it was slight. In other words there was no serious disturbance observed in the pulmonary function before the load-adding exercise.

INFLUENCE ON THE CARDIAC FUNCTION

Tagawa and Numata made an electrocardiographic study of cardiac function before operation and for six months after operation. In the experiment, standard limb leads and unipolar chest leads were used. The changes in the cardiac axis had a tendency to show a right axis deviation immediately after operation. The inverted T_1 and the flatness of T_1 wave were observed. Previously a light myocardiac disturbance was observed in all of the cases (especially in V_1 –V_3 waves) and digitalis was given to 3 patients in whom recovery seemed likely. Immediately after operation, low amplitude, flattened T_3 and inverted T_3 and depressed S –T_3, probably caused by hypoxia, were observed,

TABLE III POSTOPERATIVE VENTILATORY FUNCTION AT REST AND DURING EXERCISE

	(A) BILATERAL LOBECTOMY		(B) UNILATERAL LOBECTOMY CONTRALATERAL SEG RESECTION		(C) BILATERAL SEGMENTAL RESECTION	
	AT REST	DURING EXERCISE	AT REST	DURING EXERCISE	AT REST	DURING EXERCIS
O_2 consumption (c.c.)	275	310	265	293	292	304
Minute ventilation (L.)	9.9	11.5	10.0	10.4	11.5	12.0
Respiratory rate	13	17	16	16	17	19
Ventilation equivalent	3.7	3.9	3.7	3.6	4.0	4.2
Vital capacity (c.c.)	1,850	1,825	2,568	2,565	2,670	2,645
Expiratory reserve volume (c.c.)	715	670	793	817	788	805
Tidal volume (c.c.)	560	500	650	674	603	583
Inspiratory reserve volume (c.c.)	635	660	1,125	1,071	1,280	1,258
Maximum breathing capacity (L.)	44.0	53.0	63.1	72.6	63.1	65.2
Pulse rate	83	87	101	105	76	87
Air velocity index	1.1	1.1	1.0	1.2	0.9	1.0

but the patients recovered nearly to normal within four weeks. Six mont postoperatively, the readings were improved over what they had been befor the operation. Immediately after operation, changes by paradoxical moveme were observed, but no disturbance by sinus or auriculoventricular blocks wa observed. Six months after the operation the circulation time (arm tongu circulation time) was measured by injection of lobeline and its minimum time wa 5.6 seconds, its maximum 11.8 seconds, and its average 9.5 seconds, which we within normal limits. Six months after the operation the two step method b Master was performed and an electrocardiogram was taken before and after th exercise, there were no serious changes besides a slight tachycardia (Figs. 1 and 15).

RESULT OF THE OPERATION

The reported results (Lowell and Conklin,[11] Shumway and associates[1] a that bilateral resection was followed by no more complications than unilateral re section (Table IV).

TABLE IV POSTOPERATIVE COMPLICATION

	LOWELL AND CONKLIN (17 CASES)	SHUMWAY ET AL (15 CASES)	AUTHOR (22 CASES)
Bronchopleural fistula with empyema	2	0	0
Bronchopleural fistula without empyema	1	0	0
Empyema without fistula	0	0	1
Postoperative exacerbation	2	0	0
Contralateral pneumonia	1	0	1
Cyanosis	0	1 (tracheotomy)	0
Wound dehiscence	0	1	0
Air leak	0	1	0

Our patients were observed for eight to twenty four months after the operation, and though they need further observation, we are giving the results u to the present time. There have been no postoperative deaths or bronchia fistulas in these cases. There was one case of empyema after the secon operation but it was temporary and localized. After treatment by drainag of the thorax, intrathoracal washing, and antimicrobial therapy, the empyem receded without the need for obliterative thoracoplasty. There was one case of reactivation of the diseased focus in the contralateral side after the first operation and because of this the proposed area for resection in the contralateral side had to be enlarged. The postoperative course of the other patients was without complication. In the culture test after operation, there was only one positive case, the others were negative by smear and culture. The symptoms have receded in 10 patients and they have returned to their ordinary way of life. The remaining patients will be discharged soon to their usual pursuits.

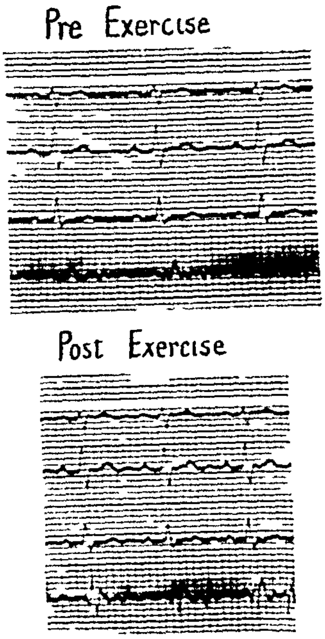


Fig. 14.

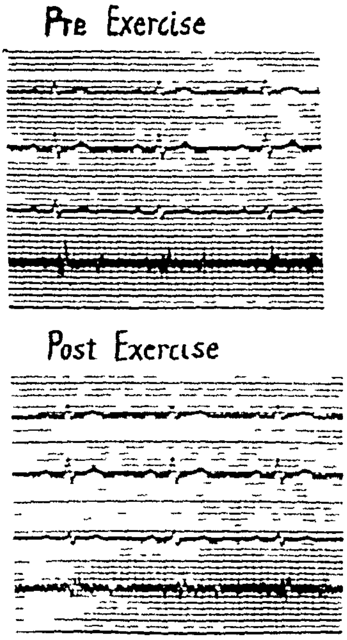


Fig. 15.

Fig. 14.—Postoperative electrocardiogram of bilateral lobectomy case before and after exercise.

Fig. 15.—Postoperative electrocardiogram of bilateral segmental resection case before and after exercise

CASE REPORTS

Case 1.—Bilateral segmental resection. The patient was a 35-year-old man who became ill during the spring of 1953. The preoperative chest x-ray finding is shown in Fig. 16 and dark shadows are seen in both of the upper pulmonary fields. A cavity is seen in the left side. Tuberculous bacterium in the sputum was positive, showing G-6. Vital capacity was 3,900 c.c. Breath-holding time, 60 seconds. Left segmental resection (S_{1+2}, S_3) was performed in September, 1953, and 3 months later a right segmental resection (S_1) was done. The postoperative course was satisfactory and without dyspnea. The postoperative x-ray finding is shown in Fig. 17. Sputum culture gave negative findings. Vital capacity at the time of discharge (8 months later) was 3,600 c.c. Recently during the patient's visit to the outpatient clinic (1 year, 5 months after operation) the vital capacity was 4,100 c.c., which was greater than that before the operation. The man, who is a woodman, is able to go into the forest to cut wood without difficulty.

Case 2.—Unilateral segmental resection with contralateral lobectomy. The patient was a 25-year-old man in whom symptoms appeared in the spring of 1952. Preoperative x-ray finding is shown in Fig. 18 and a cavity is seen on the right and a localized dark shadow to the left. As the sputum was positive for tuberculous bacterium, the patient desired operation. In November, 1953, a right upper lobectomy with thoracoplasty was performed and 3 months later a segmental resection was done on the left side, with a satisfactory course. Eleven months after operation, the patient was discharged and is now performing his everyday work without trouble. The postoperative x-ray is shown in Fig. 19, and the present vital capacity is 3,300 c.c. (600 c.c. less than preoperatively). Breath-holding time is 56 seconds.

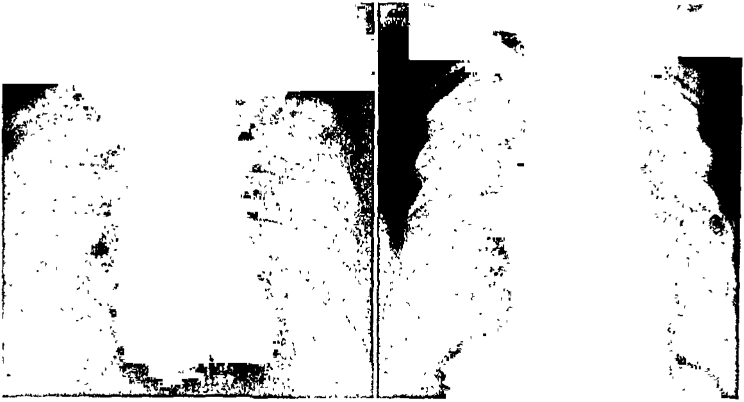


Fig. 16. Fig. 17.

Figs. 16 and 17.—Case 1. Roentgenograms of a patient who had a bilateral segmental resection. Fig. 16, preoperative; Fig. 17, postoperative.

CASE 3.—Bilateral lobectomy. The patient was a 27-year-old man who developed tuberculosis in 1949. Extensive foci were found in both lung areas and after four years of rest and chemotherapy, the sputa tests were still positive and the patient desired operation.

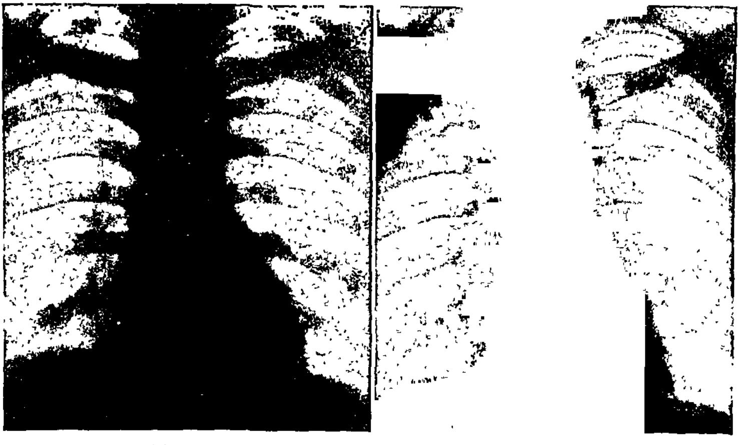


Fig. 18. Fig. 19.

Figs. 18 and 19.—Case 2. Roentgenograms of a patient who had a unilateral segmental resection with contralateral lobectom [illegible] preoperative [illegible] postoperat

Preoperative vital capacity was 2,350 c.c. Roentgenogram of the chest is shown in Fig. 20. There was a severe deviation of the mediastinum to the left, the lung appeared contracted, and cavities were seen in both lungs. The patient was not a good candidate for operation, but one was performed as a last resort. Thoracoplasty was not possible because of the pulmonary function, so consultation was held to decide what should be done for the contralateral side after performance of a lobectomy on the unilateral side. Fortunately, the vital capacity was 700 c.c. by bronchospirometry postoperatively on the operated side, so,

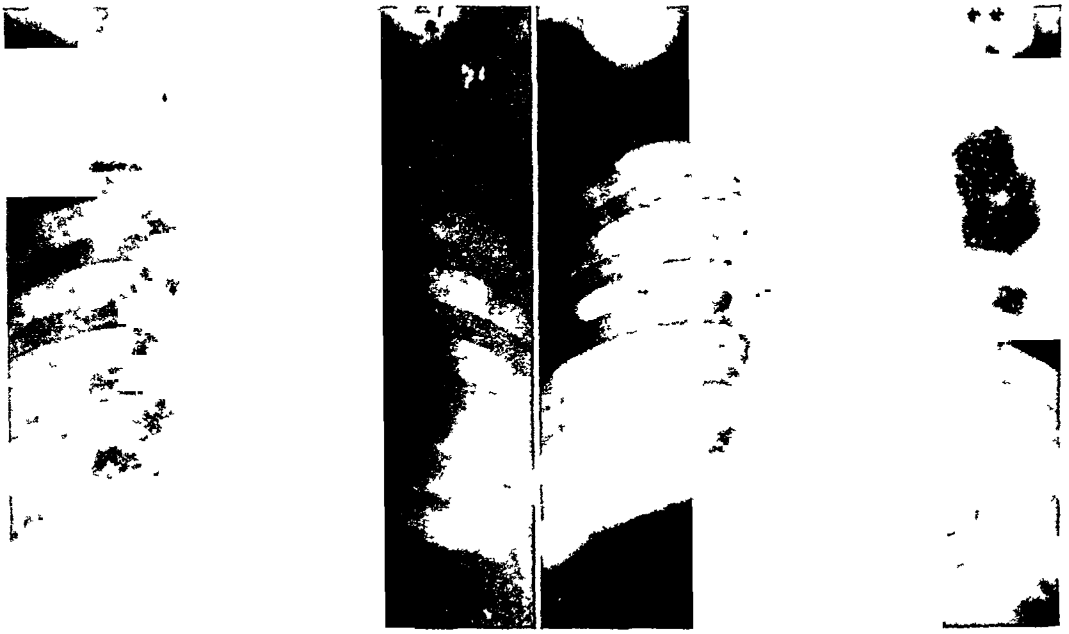


Fig. 20. Fig 21.

Figs 20 and 21.—Case 3. Roentgenograms of a patient who had a bilateral lobectomy. Fig 20, preoperative, Fig 21, postoperative

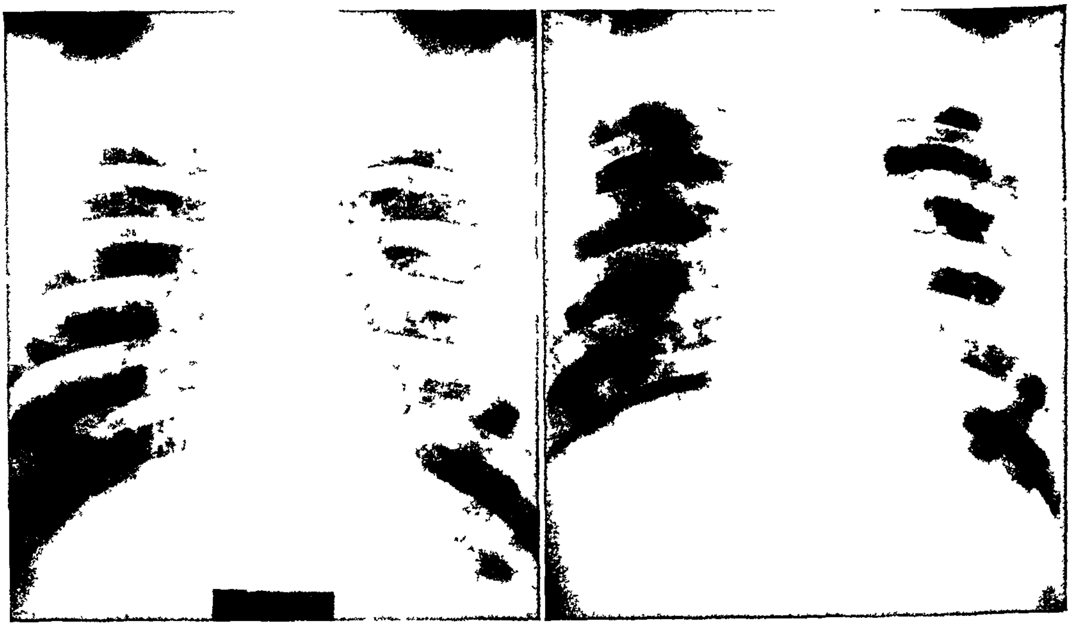


Fig. 22. Fig. 23

Figs. 22 and 23.—Roentgenograms of a patient who had a bilateral segmental resection Fig 22 shows dark shadow in the bilateral apex before operation. Fig 23 is a postoperative x-ray revealing complete re-expansion without rib resection after four months, the second operation was performed. There was no postoperative dyspnea and the patient's course was satisfactory. No tubercle bacilli were seen in the culture or the smear. The vital capacity after both operations was 2,000 c c. and the breath holding time was 30 seconds The postoperative x-ray is shown in Fig 21. No thoracoplasty was performed The patient was discharged a year later and is now working as a clerk.

CONCLUSION

We have reported on 22 cases of bilateral resection eight months after operation

Judging by the postoperative results in these patients and their pulmonary and cardiac functions, if the candidates for operation are picked with care, bilateral resection seems to be successful in returning these patients to their ordinary way of life.

The largest resected area in one case was 8 segments of both upper lobes and the left S6 and 9 segments of both lower lobes. There were no postoperative difficulties.

After a rigid examination, if this method seems adaptable to the case, indication of pulmonary resection can be extended to a greater degree. This would give more hope to these patients.

In closing, we would like to stress the importance of bronchospirometric examination in this type of operation.

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A TECHNIQUE OF PERICARDIECTOMY

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A TECHNIQUE of pericardiectomy is presented which has been used successfully in a number of cases of constrictive pericarditis at this hospital during the past four years.

The outstanding contribution and greatest impetus to us was the transsternal incision described by Julian Johnson. In using and having seen used various other sternal splitting and transcostal incisions on previous cases, great difficulty was encountered due to lack of exposure, particularly in decortication of structures of the right side of the heart.

We used the transsternal approach for the first time in July, 1951, and have become enthusiastic about the incision. The exposure is adequate for extensive decortication of all or any portion of the heart without recourse to second-stage procedures. With an anesthetist experienced in endotracheal anesthesia, this approach is as safe as any other type of incision (Figs. 1, 2, and 3).

The closure is surprisingly easy. The sternum is readily approximated with wire sutures through bone only. As a precaution, in 2 cases, pericostal wire sutures were also used, but this is not usually necessary. In 1 case, three pericostal wire sutures were removed postoperatively due to nodular reaction about the sutures.

The etiology and diagnosis of constrictive pericarditis have been described thoroughly by others, particularly in the classic thesis of Holman. Our preoperative management was similar, tuberculosis being suspected in all cases. It should be emphasized that postoperative treatment for tuberculosis should be intensive and extend over at least a one-year period. One of our first patients had received treatment for only six months postoperatively, and two years after decortication had a reactivation of pulmonary tuberculosis. After intensive therapy, the tuberculosis is arrested again, and the patient has been back at work for one year.

TECHNIQUE

The patient is placed in the supine position, and an endotracheal tube is passed under a combination of local and Pentothal anesthesia. Large-sized polyethylene tubing or 15-gauge needles are placed in the veins of each leg for rapid administrations. A simple infusion is started in the antecubital vein for the administration of procaine or other needed medications. Fluids are kept at a minimum until the decortication is completed. Electrocardiogram

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leads are attached to both arms and legs, using eight-foot extensions. The electrocardiogram writer is kept at the head of the table under the supervision of the medical cardiologist. Continuous tracings are needed so that manipulation may be discontinued for short intervals when irregularities in the tracing occur. It is a distinct aid when decortication is done about the coronary arteries, for a current of injury is immediately seen in the tracing, even on compression only of the artery. This change will soon disappear unless permanent damage has been done.

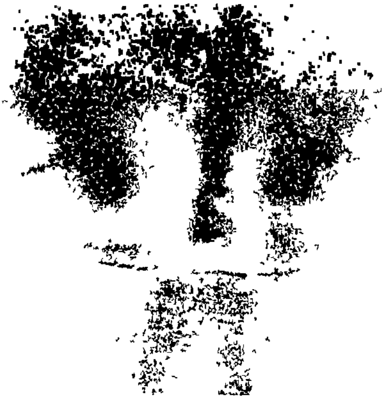


Fig. 1.

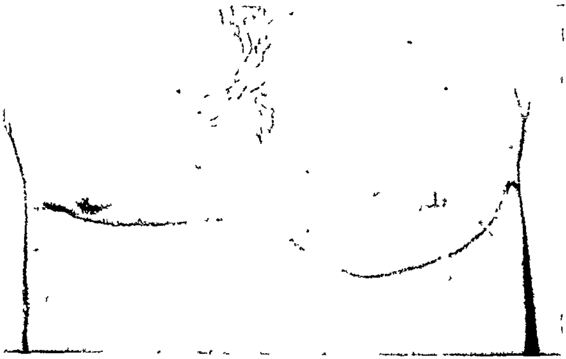


Fig. 2.

Figs 1 and 2.—Variations in the incision which may be used to meet existing conditions

After appropriate skin preparation and draping, an intercostal incision is made in the left fifth interspace from the lateral border of the pectoralis major to the sternum. The left chest and heart are explored and the diagnosis confirmed. The incision is carried over the sternum and into the right fifth interspace to the lateral margin of the right pectoralis major. The right and left internal mammary vessels are identified, ligated, and divided. The attachments of the heart to the undersurface of the sternum (the superior and inferior sternopericardial ligaments) are then dissected. A Gigli saw is passed substernally and the sternum severed at the level of the fifth interspace. The excess tissue of this ligamentous attachment of the heart is excised. This leaves a clear area varying from one to two inches in width where the process of decortication is begun.

An incision just to the left of the midline and longitudinal to it is made with a No. 15 blade knife held obliquely in this "clear" area. The left margin is picked up with a small Adson tissue forceps and a line of cleavage developed. In theory, it seems only logical first to decorticate the left ventricle to relieve pulmonary engorgement, which might occur if the right ventricle were dissected first, and to maintain a stabilized systemic pressure. In actual practice, due to ease and safety of decortication, that part of the right ventricle left of the midline is decorticated first. In no case was cardiac failure or pulmonary hypertension noted. Other observers have decorticated the *entire* right heart with similar results. The left ventricle, particularly the apex, is then decorticated before surgery of the remaining right heart. At surgery, the right ventricle and apex, the remaining left ventricle, the right atrium, inferior vena cava, superior vena cava, and left atrium have been constricted to a greater degree in the order named.

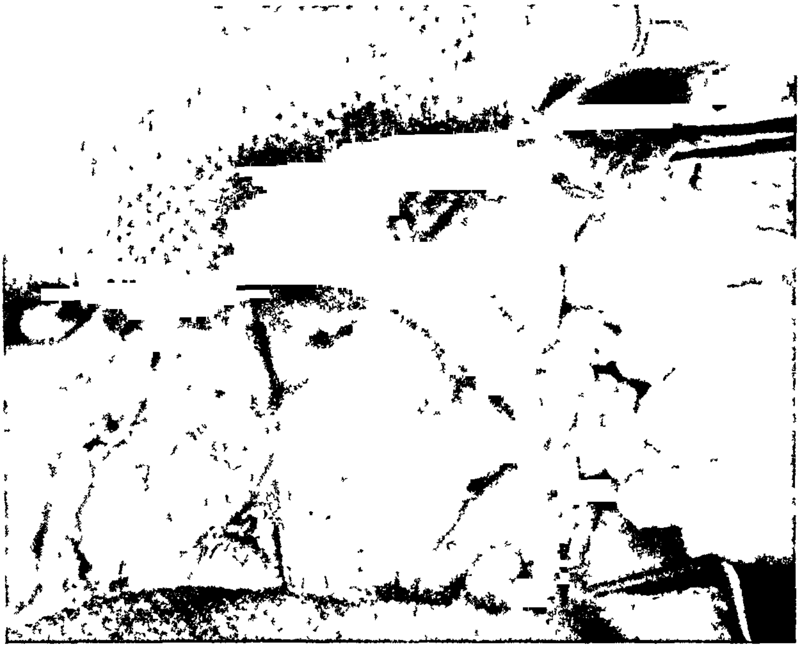


Fig. 3.—Shows wide exposure afforded by the incision, and the attachments of the superior and inferior sternopericardial ligaments.

In all cases performed, two separate and distinct layers have been found as recorded by other authors. The outer layer will separate rather easily and rapidly with finger dissection. This layer is the greatly thickened fibrous layer of the pericardium and is firmly attached, and may be fused to the central tendon of the left side of the diaphragm. It is much safer, but may be more time consuming, to free and excise this outer layer first since the outline of the coronary vessels becomes visible when it is removed. Cardiac contractions become more forceful and are depicted, in most cases, by increased amplitude of the QRS complex. The right and left heart are freed of this outer layer beyond the border of the phrenic nerves to the coronary sinus on the left and the sulcus terminalis on the right. Both phrenic nerves are freed, cutting away the rind as close to the nerves as safety will permit.

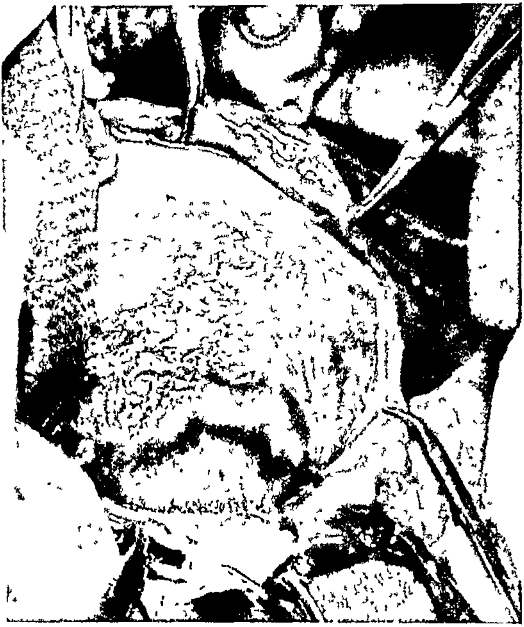


Fig 4.—Shows the two layers of the thickened pericardium and how the rind is left intact in following the advancing line of cleavage

The deep layer, though not as thick, is the true adherent and constrictive rind. It is composed of the thickened serous layer of the pericardium. This rind is carefully incised, again just to the left of the midline in the "clear" area, and the correct line of cleavage between the visceral epicardium or cardiac musculature and the rind found and developed. We have observed no fat in this area. This is probably the most difficult and tedious part of the entire procedure. The decortication continues to the left over the right ventricle with finger dissection developing the line of cleavage. Occasionally a tough band of scar tissue will have to be cut to prevent tearing into a ventricle. If the rind is left intact in following the advancing line of cleavage as described by Bigger, and others, this may be tacked down again should a ventricle be inadvertently opened (Fig. 4). The right ventricle and atrium, of course, is much thinner than the left and we entered it on one occasion. Following the recommended technique, the tear was easily closed with negligible blood loss.

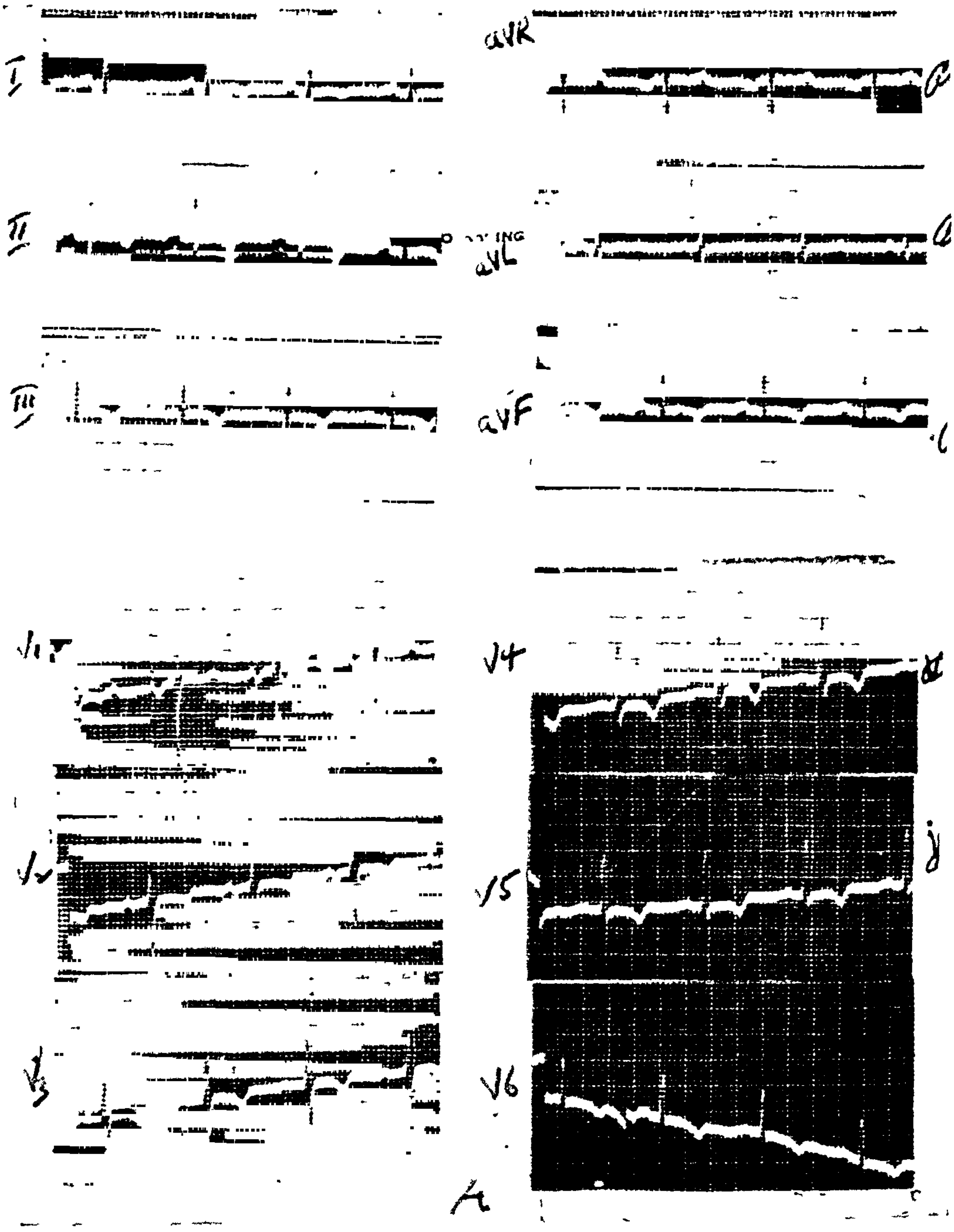


Fig 5A.—Electrocardiogram before surgery

This patient, having been bedridden for four months prior to surgery, now drives a large truck for a living, so it is presumed there have been no untoward effects. Electrocardiogram tracings of this patient show only changes consistent *with diaphragmatic ischemia (Figs. 5A and 5B).*

As the process of decortication continues, when the interventricular branch of the left coronary is reached, extreme care should be taken because the inner layer may be fused to the wall of the blood vessel. This is particularly true when, after releasing the apex, the posterior and inferior surface of the left ventricle is freed, for it is here that the marginal branch may be damaged. It may be necessary to leave a small band of rind in this area. Decortication continues to the margin of the coronary sinus posteriorly. In our cases, the left auricle has been relatively free of any constriction.

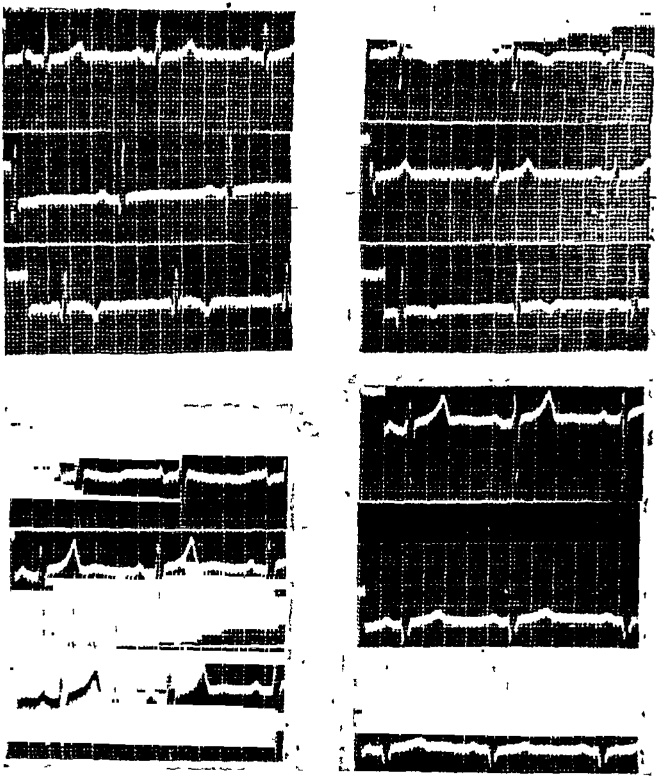


Fig 5B.—Electrocardiogram after surgery. Tracings after surgery show mild changes consistent with diaphragmatic ischemia.

The right border of the inner layer is then picked up and the rest of the right ventricle, right atrium and its appendage is freed to the sulcus terminalis, freeing the superior vena cava and the conus, superiorly.

The inferior vena cava is decorticated last. The rind is tough in this region, and the vessel is friable anteriorly because it is here that the inner layer of the pericardium may be confluent with the wall. Posteriorly, we have found the vessel to be relatively free. This completes the pericardial decortication (Fig. 6).

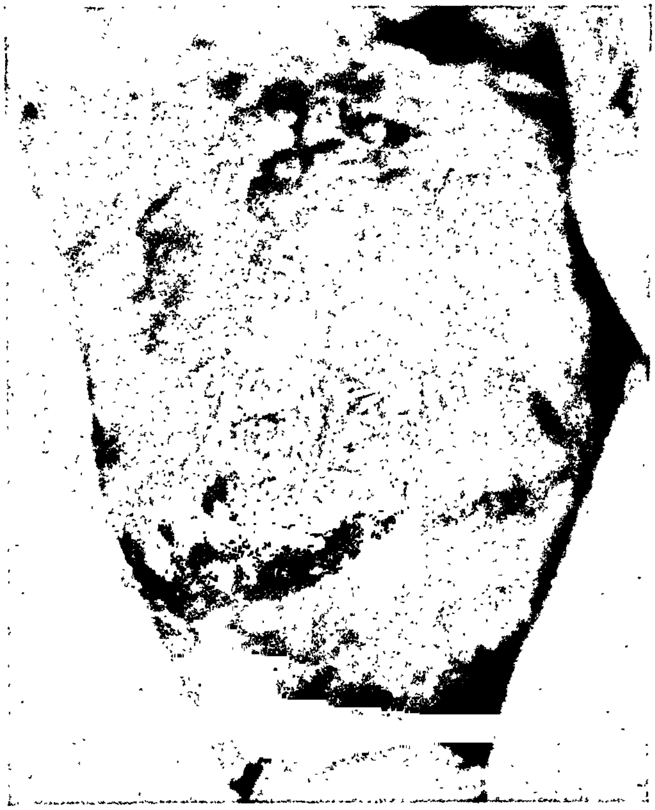
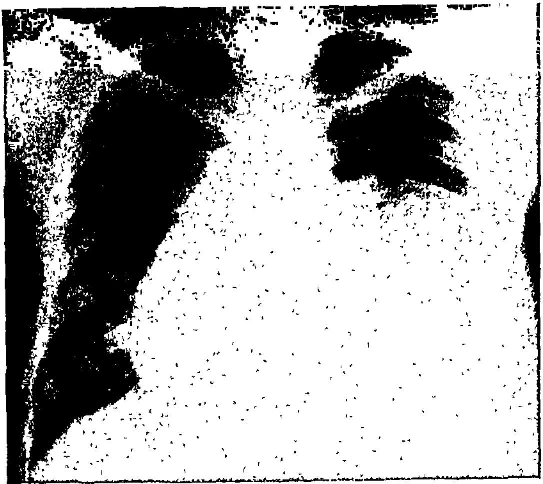


Fig. 6.—Heart at completion of decortication.

In closing the incision, two holes are drilled through both cut margins of the sternal bone and No. 26 wire passed through to the corresponding side. Interrupted sutures are then taken through the pleura and intercostal muscles, all sutures being held. Appropriate size catheters are then brought out the lateral margins of the incision to drain the right and left chest, respectively. The catheters are connected in the usual fashion to three-way, pressure regulated, underwater-seal bottles and to Gonco suction apparatus.

The rib approximator on each side brings the wound edges together easily and all sutures are tied beginning with the sternal wires. The rest of the incision is closed in layers with interrupted sutures. To complete the procedure, x-rays of the chest are taken while the patient is still on the table to be sure both lungs have been re-expanded. The patient is then moved to the recovery ward for postoperative care (Figs. 7 through 11).

Fig. 7.



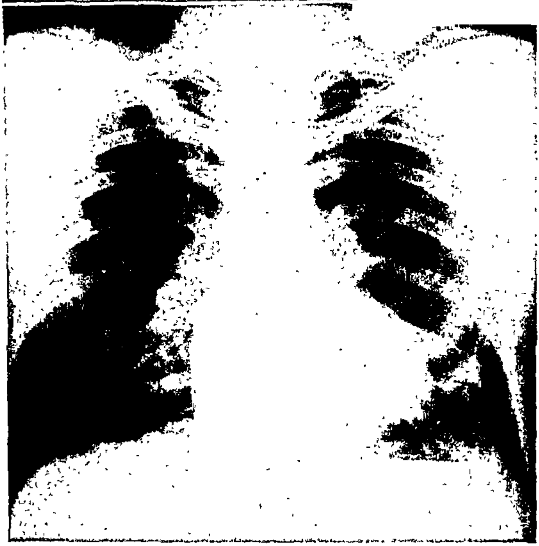


Fig. 8.

Fig. 7.—Preoperative film of chest in a case of tuberculous pericarditis with ascites and pleural effusion.

Fig. 8.—Postoperative film of same patient taken one year after surgery.

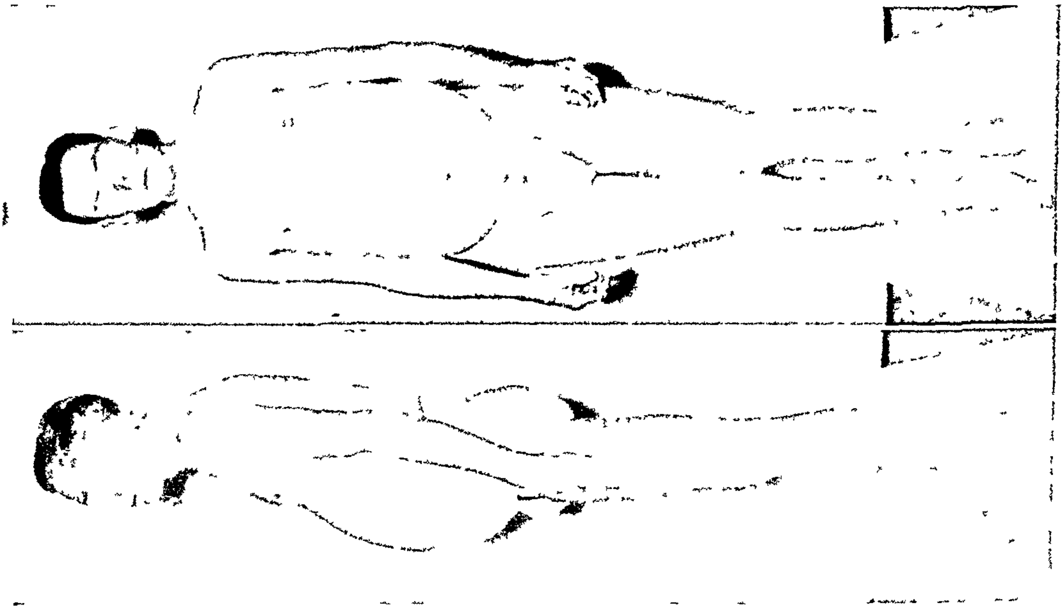


Fig. 9.

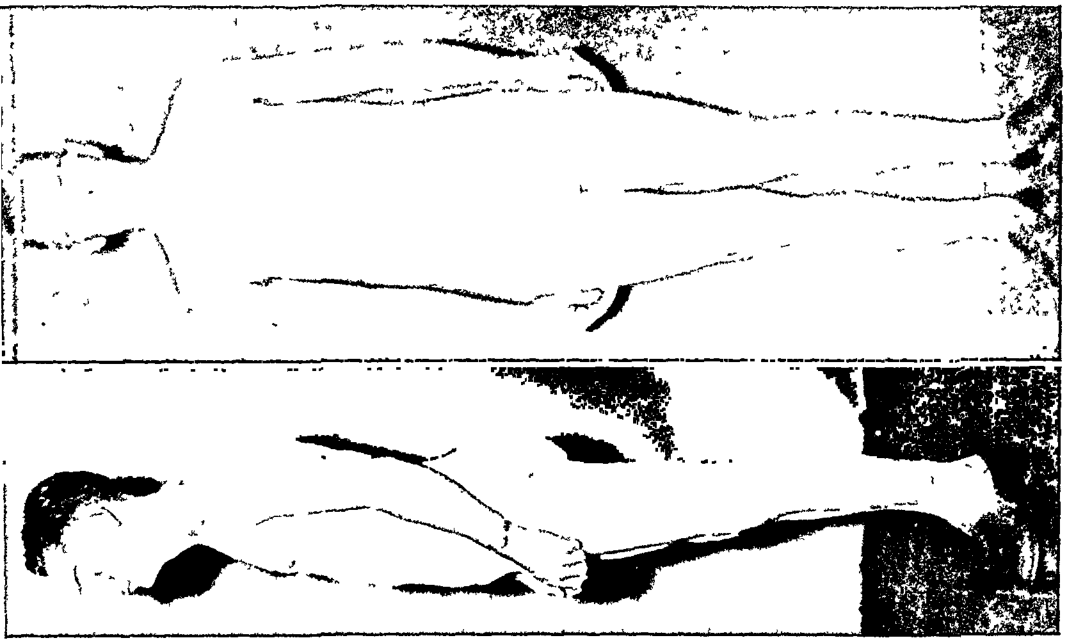


Fig. 10.

Fig. 9.—Preoperative views of patient with tuberculous pericarditis

Fig 10.—Postoperative films of same patient one and one-half years after decortication

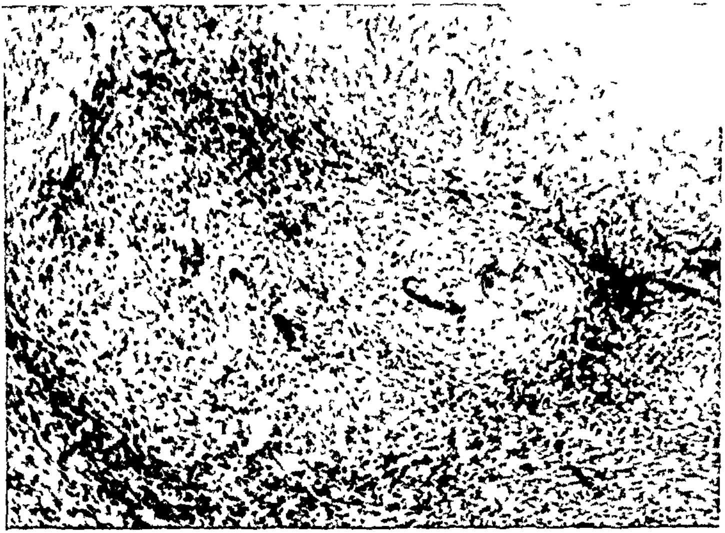


Fig 11.—Shows typical tubercle formation in a case of constrictive pericarditis. In this case, tubercle bacilli could not be cultured or found by acid-fast stain. (Tubercle bacilli were found by acid-fast stain in 3 of 10 cases—none could be cultured.)

SUMMARY

1. A technique of pericardiectomy is presented.

2. Decortication is begun in the "clear" area of the heart (the area of the attachments of the superior and inferior sternopericardial ligaments).

3. Rind is left intact in following the advancing line of cleavage as described by Bigger and others.

4. The apex and that portion of the right ventricle to the left of the midline is decorticated first, followed by the left ventricle, and the remaining right heart and great veins.

5. The inferior vena cava is decorticated last.

6. Closure is easy.

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THE SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS IN MENTAL PATIENTS

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INTRODUCTION

THE importance of tuberculosis in mental institutions was recognized in Minnesota as early as 1897 and separate facilities for the isolation of mental patients with tuberculosis were provided at two of the State institutions during the first decade of the twentieth century. However, it was not until 1934 that an effective program for control of this disease in State institutions actually began.[11] That year, Dr. Herbert A. Burns surveyed, by tuberculin test and x-ray examination, all inmates and personnel of the nine State institutions for the mentally ill in Minnesota. His study,[7] showing an inordinately high incidence of tuberculosis among both the patients and those caring for them, led eventually to the establishment of a separate tuberculosis hospital for all mentally ill persons, the Herbert A. Burns Memorial Hospital, which was opened as part of the Anoka State Hospital in 1950.

Since July, 1952, surgical treatment at the Burns Hospital has been carried out in conjunction with the Department of Surgery of The University of Minnesota. Some of our earlier experience in treating these patients has been reported previously.[8, 9, 13] This article is a current summary of our accomplishments in this special group of patients.

The achievement to be realized in treating these patients is less than that attained in patients without mental disease. With few exceptions, these individuals are permanent residents of mental institutions. Nevertheless, most of those who have had pulmonary resections can be removed from the isolation and confinement necessary in a tuberculosis hospital. Furthermore, they no longer threaten the health of other mental patients, the hospital attendants and, indirectly, the population at large.

To achieve these ends we have felt that it is best to remove surgically all foci which might with time reactivate. Before discharge from the Tuberculosis Unit, it is important that the sputum and gastric cultures of treated patients be consistently negative for in mental hospitals the disease is apt to be contagious. Based on this line of reasoning [illegible] criteria for operation have been more liberal than those usually employed [illegible] patients without mental disease. We have

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operated upon all patients in relatively good physical condition if the disease was quiescent and not so widely spread as to prohibit the removal of all significant caseous and cavitary foci.

CLINICAL MATERIAL

To March 1, 1955, 187 patients have had pulmonary resections. All patients have received at least four months of antibiotic therapy prior to surgery, and a total course, both pre- and postoperatively, of at least twelve months. One gram of streptomycin twice a week and 200 mg. of isoniazid daily have been used routinely with para-aminosalicylic acid as a substitute or additional drug in some cases. Bed rest has played little or no part in treatment chiefly because enforced bed rest, among the mentally ill, is not practical.

Age and Sex.—As shown in Table I, our patients have been older on the average (46.6 years) than those in any reported series of patients treated by pulmonary resection.[4, 5, 7, 10, 12] Approximately 40 per cent of our patients were in the fifth decade of life or older. There was a preponderance of men over women in the ratio of 1.35 to 1. Thus, it seems that, at least among mental patients, tuberculosis is characteristically a disease of aging men.

TABLE I. AGE DISTRIBUTION BY DECADES OF PATIENTS UNDERGOING RESECTIONS FOR TUBERCULOSIS
(RANGE: 18-73 YEARS)

AGE	NUMBER OF PATIENTS
11-20	1
21-30	20
31-40	37
41-50	50
51-60	56
61-70	21
71-80	2
Total	187

Mortality Rates and Complications.—There were 16 deaths, early and late, in the entire group, an over-all mortality rate of 8.5 per cent. The causes of death are given in Table II. Respiratory complications in the postoperative period have been responsible for the greatest number of deaths. This reflects the difficulty of postoperative management encountered in uncooperative patients. Tracheotomy has been utilized twelve times after operation but despite this and the frequent use of bronchoscopy, pneumonia and atelectasis have been the most common causes of death.

TABLE II. CAUSES OF DEATH IN PATIENTS UNDERGOING PULMONARY RESECTIONS

Atelectasis and pneumonia	9
Empyema, bronchopleural fistula	2
Massive pulmonary hemorrhage	1
Pulmonary embolism	1
Mediastinal flutter (flail chest)	1
Cardiac arrest	1
Cause unknown	1
Total	16

Bronchopleural fistula has also been a troublesome late postsurgical complication It occurred in 13 patients following unilateral resection and in 1 patient following both sides of a staged bilateral operation Two deaths have occurred among the patients who have had these fifteen bronchopleural fistulas, a mortality rate for this complication of 13 3 per cent One of these deaths followed an unsuccessful attempt at closure by a muscle flap transplant and the other death was due to empyema, in this case the bronchopleural fistula was not recognized until autopsy Among the 13 remaining patients with fistulas thoracoplasty plus catheter drainage was a successful treatment in all except 3 These 3 required a muscle flap operation for closure after a thoracoplasty had proved inadequate

Type of Operation—Unilateral resections were performed on 146 patients and bilateral operations were completed in 41 others (Table III) In the table it will be noted that the mortality rate has been highest among patients who have had a unilateral lobectomy The explanation of this is not apparent for the causes of death in this group have not been materially different from the causes of death after other pulmonary resections Unilateral segmental resection has been followed by a more acceptable mortality rate of 3 1 per cent Still many of the patients in this group have had multiple segments removed and the incidence of complications has been as high as it was following lobectomy Indeed, all bronchopleural fistulas except one occurred after segmental resection

TABLE III PULMONARY RESECTIONS FOR TUBERCULOSIS

TYPE OF OPERATION	NUMBER OF PATIENTS	DEATHS
I Unilateral Operations		
Lobectomy	29	10
Wedge	9	1
Pneumonectomy	12	2
Segmental	96	3
II Bilateral Operations		
Staged	26	0
Simultaneous	15	0
Total	187	16 (8 5%)

Bilateral operations were performed in 26 patients as staged procedures and in 15 patients the disease in both lungs has been extirpated at one operation In the group with staged bilateral resections the only significant complications were bronchopleural fistula which occurred in 1 patient and residual active endobronchial disease which persists in another No deaths have occurred among the patients who have had staged bilateral operations

The technique of the simultaneous bilateral resection has been described previously[9, 13] Briefly, the chest is opened transsternally in the fourth interspace anteriorly and both pleural cavities are entered The indicated resections are carried out first on one side and then on the other as the table is tilted from side to side As a rule this operation has been reserved for patients who require no more than segmental resections bilaterally though 1 patient has had a lobect[illegible] side and a segmental resection on the other With this techni[illegible]ation *can accomplish as much as two thoracotomies and the* total p[illegible]tment has been greatly shortened Sternal separation has been a troublesome postoperative complication but there have been no deaths in the group. There have, however, been two deaths in patients with exploratory operations through the transverse sternotomy incision. Both of these were relatively poor risk patients with bilateral disease in whom the operation was performed with the idea of resecting both sides if it seemed reasonable at the time of surgery. Because of the extent of the disease and the general response of the patients to the operation only one side was resected in each case. Neither died of complications due to the type of incision used. One died of pulmonary embolism while the other died of atelectasis and pneumonia, both on the thirteenth postoperative day.

Thoracoplasty has been employed only as an auxiliary operation. In all, 45 thoracoplasties have been done. On three occasions, extensive, uncontrolled unilateral disease was treated first by thoracoplasty and subsequently by pneumonectomy. Simultaneous thoracoplasty was carried out fourteen times with pulmonary resection and thoracoplasty was added as a second operation to reduce the size of the hemithorax, twenty-eight times. As described above, thirteen of these late operations were performed for bronchopleural fistula. The deaths which might possibly be related to thoracoplasty were four which occurred following lobectomy with simultaneous thoracoplasty.

Results of Treatment.—Table IV shows the present classification of the 171 surviving patients according to the 1950 standards of the National Tuberculosis Association. Of the 8 patients who have active disease, 5 have had persistent activity after unilateral segmental resection, 1 patient has contralateral active disease after pneumonectomy while another developed contralateral spread after lobectomy. The eighth patient has endobronchial tuberculosis after a bilateral staged segmental resection.

TABLE IV. PRESENT STATUS OF DISEASE IN PATIENTS TREATED FOR TUBERCULOSIS BY PULMONARY RESECTION

CLASSIFICATION	NUMBER
Inactive	77
Arrested	86
Active	8
Total	171

Up to March 1, 1955, 64 patients have been discharged from the tuberculosis unit to other mental hospitals, throughout the State. In each instance, all the disease evident on chest x-ray, except perhaps linear fibrotic scars, has been removed and at least six gastric washings, taken in series, have been negative and none have been positive by culture. These discharged patients are followed by chest x-ray at three-month intervals, and three gastric cultures will be taken every six months for the first two years. The frequency of bacteriologic studies thereafter will depend on the clinical and x-ray findings.

DISCUSSION

Although we have used pulmonary resection more frequently than others who have discussed the treatment of tuberculosis in patients with mental dis-case,[1, 3, 6, 14, 15, 16] we believe that the results achieved have justified undertaking such a program. Only 8 patients have experienced late spread or reactivation. On the other hand, we have already been able to return 64 patients to nontuberculosis institutions with the expectancy that they will remain free of their disease, and there are, in addition, an equal number of operated patients who await only bacteriologic confirmation of their apparently arrested state. In each instance, there remains no visible pulmonary or other focus of tuberculosis and of the 64 who have been discharged each had at least six gastric washings that have been negative by culture for tubercle bacilli. With the collapse measures that have been performed by others for mental patients with tuberculosis, it is improbable that results this good would have been obtained.

Not all patients at the Burns Hospital can be treated successfully by surgery. Approximately 100, out of a total population of 420 patients, are too old or physically unfit for operation but at the present time there are at least 90 patients awaiting surgery who can be operated upon with the same chance for success as has been achieved among those reviewed here. In addition, newly diagnosed cases are sent to the Burns Hospital so that there will doubtless be a significant number of new patients at least for some time. Most of these new patients will doubtless be candidates for surgery and in time the number of mental patients with active tuberculosis in Minnesota should be reduced to a small irretrievable minimum. Tuberculosis in mental hospitals can be controlled.

The existence of bilateral disease does not constitute a barrier to successful surgical treatment. Except in patients with widely scattered disease in both lungs, bilateral operation may be carried out with reasonable expectancy for success. Those with bilateral apical disease can be treated surgically at one operation.

SUMMARY

1. One hundred and eighty-seven mental patients with pulmonary tuberculosis have had pulmonary resections performed. Of these, 146 patients underwent unilateral operations and 41 have had bilateral resections performed. Fifteen patients have undergone bilateral simultaneous resections.

2. The over-all mortality rate was 8.5 per cent. The difficulties encountered in postoperative management of uncooperative patients have contributed to this mortality rate.

3. Late results of treatment have been good. Of 171 patients who have survived, only 8 can be classified as having active tuberculosis at the present time.

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RECURRENT CARCINOMA IN THE BRONCHIAL STUMP*

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DURING the past twenty years the medical profession has noted a marked increase in its responsibility toward the problem of bronchogenic carcinoma. The change has occurred because of the rising incidence of the disease and the concomitant development of safe and practical thoracic surgery. Today the diagnosis of pulmonary cancer demands an evaluation of operability. The latter, dependent on many factors, may still be questionable at the time of exploration. There may be lymphatic, pleural or chest wall involvement the complete excision of which remains in doubt even after the resection has been carried out. In addition, the extension of the primary growth in the region of the carina and main bronchi is often difficult to evaluate.

The present study of recurrent carcinoma in the bronchial stump was undertaken with the preceding considerations in mind. It seemed of practical value to determine if neoplastic tissue had been left behind in the remaining bronchial segment, whether the tumor had actually been inoperable from the bronchoscopic standpoint or if other factors might be of importance in the etiology of this type of neoplastic recurrence. Also, the clinical aspects of recurrent carcinoma in the bronchial stump appeared worthy of study.

At the outset it should be noted that the term "recurrent" is used in its unrestricted sense throughout this paper. It will be apparent from an analysis of results of this study that whereas some of the cases are not examples of recurrent carcinoma in the limited connotation of that word, there are others in which the term is strictly applicable. The similar clinical pattern of all the cases justifies the common grouping.

METHOD OF STUDY

Six hundred and thirty one patients underwent resection, either lobectomy or pneumonectomy, for carcinoma at the Mayo Clinic prior to September, 1952. In this group there were 18 in whom recurrence of carcinoma in the remaining bronchial stump subsequently developed. These latter cases form the basis of this report. They were studied from the clinical standpoint with

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regard to the symptoms, signs, and diagnosis of recurrence and pathologically, with regard to the extent of the lesion at the time of pulmonary resection.

The clinical histories and surgical records were reviewed. In addition, a study was made of each formalin-preserved operative specimen. The presence or absence of lymph-node involvement was noted. In the gross specimens the distance was measured from the proximal end of the severed bronchus to the nearest visible tumor. Then serial longitudinal blocks were cut from the entire bronchial circumference adjacent to the proximal limits of the tumors. From these blocks, hematoxylin and eosin stained preparations were studied for evidence of microscopic extension of the bronchogenic carcinoma in the mucosa or submucous layers toward the proximal end of the resected bronchus. Particular attention was paid to cellular detail in the bronchial mucous membrane.

RESULTS OF THE STUDY

With few exceptions the clinical pattern in this series was similar for all cases. After resection of a bronchogenic carcinoma these patients returned to the clinic at intervals varying from 5 weeks to 24 months with either cough or hemoptysis or both. In all but 3 cases the recurrent tumor was visualized bronchoscopically and confirmed by biopsy or smear. The diagnosis in 1 case was established by cytologic study of secretions aspirated directly from the bronchial stump and in 2 cases by the finding of malignant cells in the sputum.

The status of the patient up to the time when this paper was written is known in thirteen instances. Of the 11 patients known to be dead, 10 succumbed to the cancer at intervals varying from 3 to 28 months following pulmonary resection. One patient died, apparently of cardiac failure and pulmonary edema, following a transurethral prostatectomy done elsewhere 26 months after pneumonectomy. One patient is known to be alive and asymptomatic 30 months after pneumonectomy and 6 months following diagnosis of bronchial-stump recurrence and treatment with deep roentgen rays and locally implanted radon seeds. One patient is living but in failing health 16 months after resection.

In all instances the tumors were high-grade (Broders) squamous-cell carcinomas. Most of the primary tumors were relatively large, the average being 3.8 cm. in the largest dimension. The size, however, varied from 1.5 cm. to 6.0 cm. in diameter. Lymph nodes were involved by metastasis in 12 of the cases.

On the basis of pathologic findings in the proximal resected bronchus, the cases in this series have been placed in five groups.

Group 1.—This group consists of 5 cases (Table I) in which examination revealed carcinoma at the proximal end of the resected main bronchus. In addition to the bronchial involvement metastatic hilar lymph nodes were present in 4 of the 5 cases. The tumor was on the right side in 3 cases and on the left in 2. Total pneumonectomy and complete hilar-node dissection were carried out in each instance. Three of the operations, however, were considered at the time of surgery to be merely palliative either because of gross involvement of lymphatic and mediastinal structures or because it became evident as the operation progressed that the main bronchus was involved so near the carina that resection with an adequate margin was impossible.

Three of the five tumors in this group were visible bronchoscopically prior to resection. Biopsy via the bronchoscope produced positive results in 2 of these 3 cases. In the other 2 cases there were bronchoscopic findings, only in retrospect, suggestive of tumor. In one of these biopsy was not done and in the other results of biopsy were negative.

TABLE I. SUMMARY OF DATA FOR CASES IN GROUP 1

CASE	PREOPERATIVE BRONCHOSCOPY	OPERATION	CONSIDERED PALLIATIVE?*	LYMPH-NODE INVOLVEMENT	INTERVAL: RESECTION TO RECURRENCE (MONTHS)	RECURRENCE DIAGNOSED BY:	INTERVAL: RESECTION TO DEATH (MONTHS)
1	Granulations in left upper lobe Biopsy not done	Left pneumonectomy	No	Yes	7	Bronchial secretions	23
2	Tumor in left main bronchus Biopsy negative	Left pneumonectomy	Yes	Yes	4½	Biopsy	9½
3	Tumor right upper lobe and main bronchi Biopsy positive	Right pneumonectomy	Yes	No	1¼	Cells in sputum	3
4	Narrowed right upper lobe bronchus Biopsy negative	Right pneumonectomy	Yes	Yes	4½	Biopsy	14
5	Tumor bifurcation right main bronchus Biopsy positive	Right pneumonectomy	No	Yes	5	Biopsy and smear	19

*Operation considered palliative at conclusion of resection.

In this group of patients the presence of residual carcinoma in the bronchial stump became apparent quite early. There was recurrent cough and hemoptysis within an average of 4.4 months postoperatively in all cases.

Complete follow-up data are available for the 5 cases. The longest interval from the onset of bronchial-stump symptoms to death was 16 months, and the shortest period was 2 months with an average of 9.3 months. The longest survival after pulmonary resection was 23 months and the shortest was 3 months with an average of 13.7 months from pneumonectomy to death. In the 3 cases for which operation was considered at the time to be merely palliative, the average survival period after resection was 8.8 months.

TABLE II. SUMMARY OF DATA FOR CASES IN GROUP 2

CASE	PREOPERATIVE BRONCHOSCOPY	OPERATION	CONSIDERED PALLIATIVE?*	LYMPH-NODE INVOLVEMENT	MARGIN OF RESECTION (CM.)	INTERVAL: RESECTION TO RECURRENCE (MONTHS)	RECURRENCE DIAGNOSED BY:	INTERVAL: RESECTION TO DEATH (MONTHS)
6	Infiltration left upper lobe bronchus Biopsy negative	Left pneumonectomy	No	Yes	1.0	13	Biopsy	Unknown
7	Lesion 1.5 cm. from carina Biopsy positive	Right pneumonectomy	No	No	1.3	10	Biopsy and smear	Unknown
8	Lesion right main bronchus Biopsy positive	Right pneumonectomy	No	No	0.3	3	Biopsy	13
9	No lesion seen Biopsy negative	Left pneumonectomy	No	No	0.6	21	Biopsy and smear	26
10	No tumor seen Biopsy not done	Right middle and lower lobectomy	Yes	Yes	1.0	20	Biopsy and cells in sputum	27
11	Tumor right lower lobe and main bronchi Biopsy positive	Right pneumonectomy	No	Yes	1.3	24	Cells in sputum and biopsy	Living 30 months after resection
12	Tumor left lower lobe bronchus Biopsy positive	Left pneumonectomy	No	Yes	1.0	10	Cells in sputum and biopsy	Living but in failing health 16 months after resection

*Operation considered palliative at conclusion of resection.

Group 2.—There were 7 cases in this study in which examination of the resected bronchi revealed an inadequate margin of resection and metaplasia of the bronchial mucous membrane proximal to the tumor (Table II) In view of the work of Griess, McDonald, and Clagett, a margin of 1.5 cm. of proximal tumor-free bronchus was considered adequate. This problem will be discussed in a later section. The finding of metaplastic epithelium in the bronchi, proximal to the primary tumors, is believed to be significant and will also be considered in more detail later.

The length of resected bronchus proximal to the tumor varied from 0 3 cm. to 1.3 cm. in the 7 cases in Group 2. There was epithelial metaplasia of the proximal tumor-free bronchus in all instances This finding is illustrated in Figs. 1 and 2, *a* and *b*. In 4 cases hilar lymph nodes were involved by metastases. The size of the primary tumors in this group varied from 1 5 cm. to 6 0 cm. in the largest dimension.

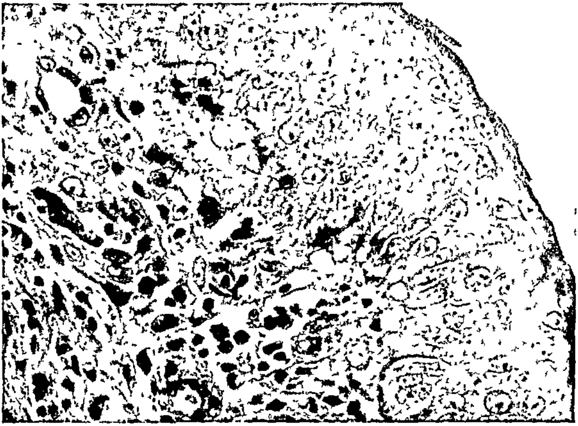


Fig 1—Longitudinal section of main bronchus proximal to the primary tumor in a Group 2 case This demonstrates marked squamous metaplasia of the bronchial epithelium in this region (hematoxylin and eosin stain, ×450)

The tumor involved the right lung in 4 cases and the left in 3. Total pneumonectomy and hilar-node dissection were performed in 6 cases. Palliative resection of the right middle and lower lobes was performed in 1 case because of extensive lymphatic involvement and fixation of the tumor in the region of the azygos vein, vena cava, and pulmonary artery.

Five of the seven tumors in this group were visible bronchoscopically prior to resection Biopsy was positive for carcinoma in four instances

The average time from resection to the onset of symptoms of bronchial-stump recurrence was somewhat over 14 months.

Follow-up data are complete for only 4 cases in this group. The interval from resection to death varied in 3 cases from 13 months to 27 months with an average of 22 months. The interval from the first symptom of stump recurrence to death averaged 7 months. The fourth patient is known to be living and asymptomatic 30 months after pneumonectomy and 6 months after stump recurrence was diagnosed and treated with deep x-rays and locally implanted radon seeds.

Group 3.—Table III summarizes data for the 2 cases in this group. The margin of bronchial resection was inadequate (less than 1.5 cm.) in each.

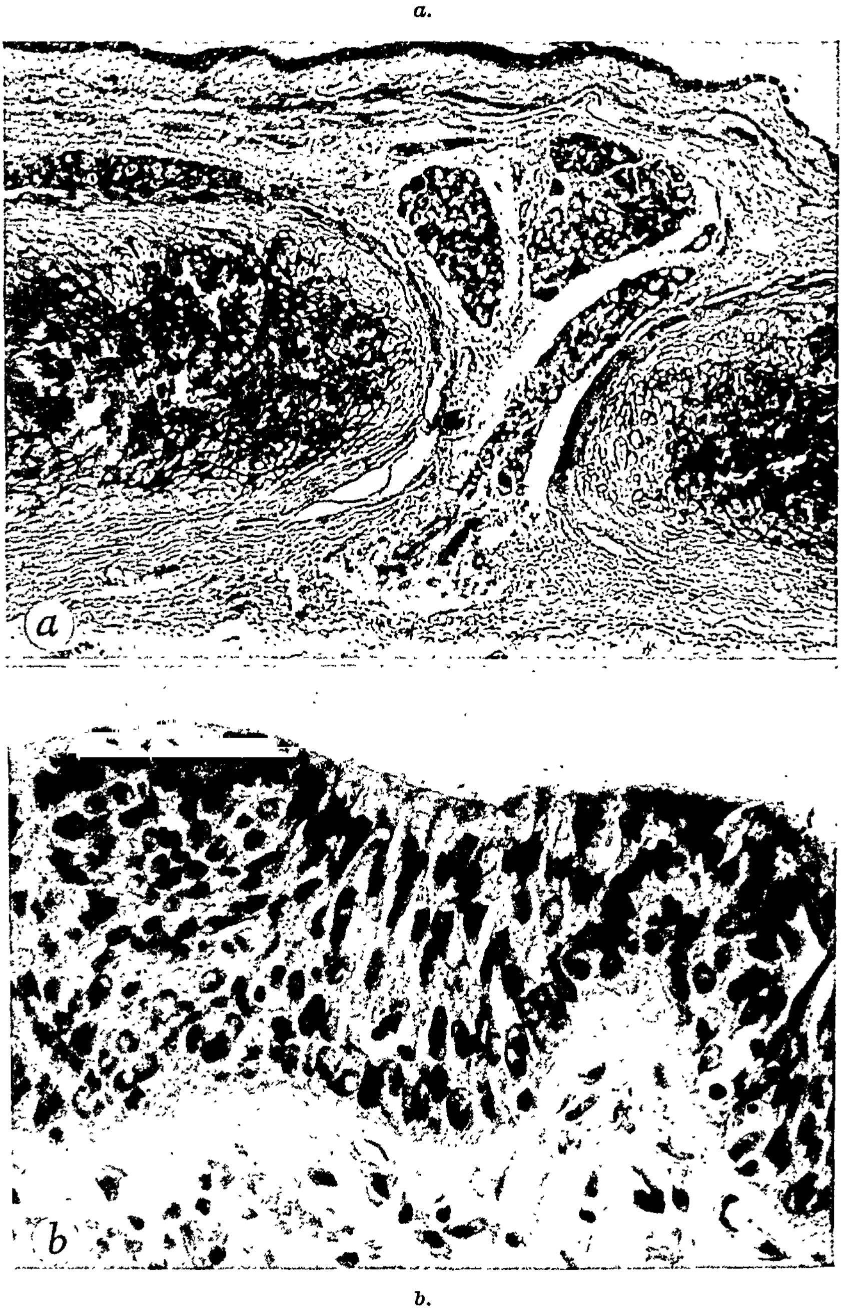


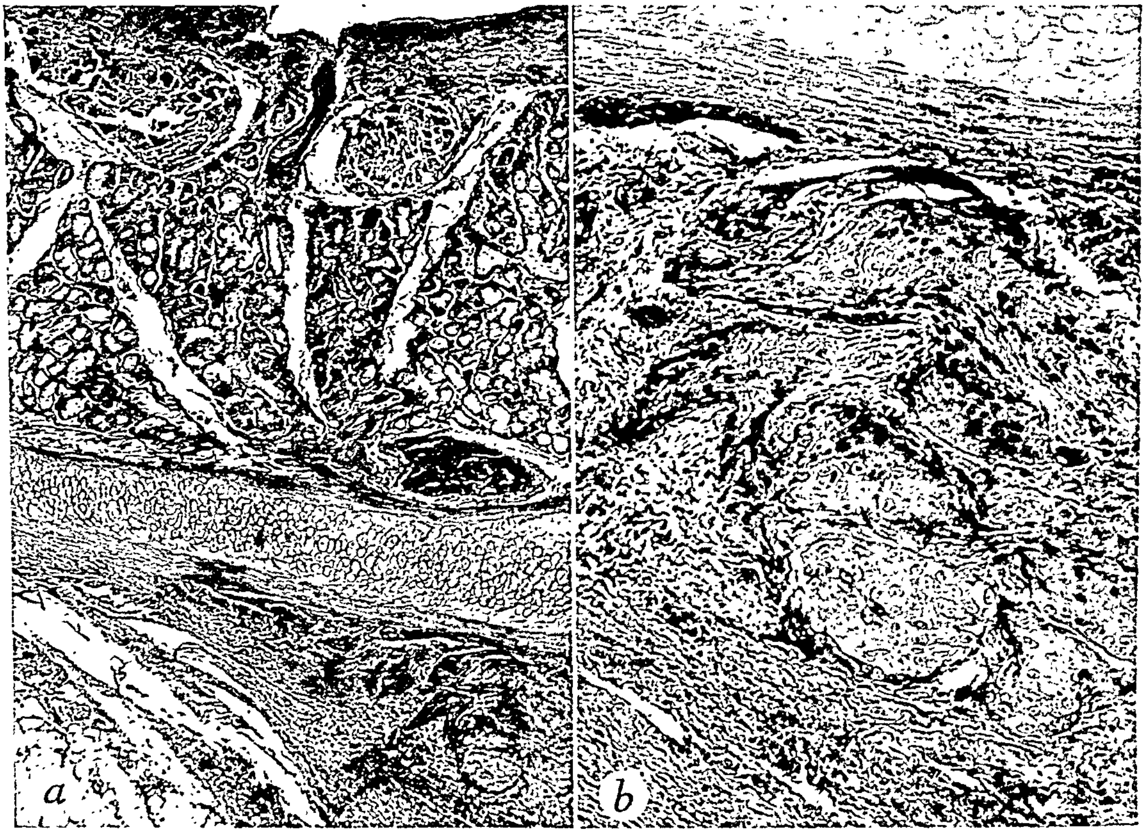
Fig. 2.—*a*. Longitudinal section at the proximal end of the resected main bronchus in another case in Group 2. The mucous membrane at the upper margin of the section is illustrated in higher magnification in *b* (hematoxylin and eosin stain; ×30). *b*. Higher magnification of the bronchial epithelium in *a*. This illustrates a rather early degree of cellular metaplasia in the bronchial mucous membrane (hematoxylin and eosin stain; ×450).

TABLE III. SUMMARY OF DATA FOR CASES IN GROUP 3

CASE	PREOPERATIVE BRONCHOSCOPY	OPERATION	CONSIDERED PALLIATIVE?*	LYMPH NODE INVOLVEMENT	MARGIN OF RESECTION (CM.)	INTERVAL RESECTION TO RECURRENCE (MONTHS)	RECURRENCE DIAGNOSED BY:	INTERVAL RESECTION TO DEATH (MONTHS)
13	Lesion superior wall left main bronchus Biopsy positive	Left pneumonectomy	No	Yes	0.75	5	Biopsy	Unknown
14	Tumor right upper lobe Biopsy positive	Right pneumonectomy	Yes	Yes	?	22	Cells in sputum and biopsy	28

*Operation considered palliative at conclusion of resection

They differ, however, from the cases in Group 2 in that no epithelial metaplasia could be demonstrated in the bronchial mucous membrane proximal to the tumor. In 1 case preoperative bronchoscopic examination revealed a squamous-cell carcinoma on the superior wall of the left main bronchus. Pneumonectomy was carried out and study of the lung showed that the tumor extended in the outer fibrous layer (Fig. 3, *a* and *b*) to within 0.75 cm. of the proximal extremity of the resected left main bronchus. One hilar lymph node was metastatically involved. Symptoms of stump recurrence appeared 5 months postoperatively, and the patient was thereafter lost to follow-up.



a. *b.*

Fig. 3.—*a.* Longitudinal section of the proximal main bronchus in a Group 3 case. This is an example of tumor extension in the submucosal layers of the bronchial wall. The area in the lower right corner represents tumor in the outer fibrous layer and is illustrated in *b* under higher magnification (hematoxylin and eosin stain; ×35). *b.* Higher magnification of the area in the lower right corner of *a* showing microscopic extension of tumor toward the proximal end of the resected bronchus in this case (hematoxylin and eosin stain; ×110).

In the second case in this group, bronchoscopy prior to operation revealed a squamous-cell carcinoma obstructing the right upper lobe bronchus. At operation the right main bronchus was resected near the carina, but on examination it was found that the tumor extended to the proximal end of the specimen. Accordingly, all the rest of the main bronchus was removed. The exact margin of resection is not known but is believed to have extended inadequately above the tumor. One hilar node was involved by carcinoma.

Symptoms of stump recurrence did not develop until 22 months postoperatively. Death occurred 28 months after the palliative resection. Necropsy is reported to have shown the tumor to be confined to the right hemithorax, mediastinum including the esophagus and heart, and the region of the carina. There were no grossly apparent distant metastases, and no involvement of the contralateral lung was noted.

Group 4.—The fourth group in this series (Table IV) consists of 3 cases in which the main bronchus was resected at least 2.5 cm. above the primary tumor. Recurrence of the malignant lesion became apparent in all cases within 6 months. The significant pathologic finding, in addition to the adequate margin of resection, was a high degree of epithelial metaplasia in the bronchial mucous membrane proximal to the tumor (Fig. 4). Hilar lymph nodes were involved by metastasis in 2 of the 3 cases. Bronchoscopic examination prior to resection produced positive evidence of carcinoma in 2 cases. In the third case, there was an area of questionable infiltration in the left main bronchus, biopsy of which was negative.

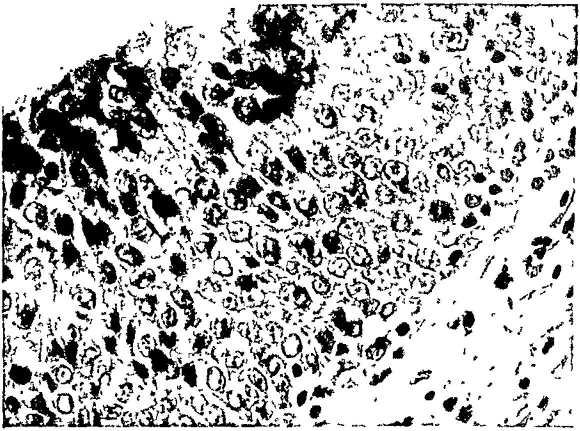


Fig. 4.—Longitudinal section of the proximal main bronchus in a Group 4 case. This is another example of cellular metaplasia at the proximal end of a resected bronchus (hematoxylin and eosin stain; ×450).

In 1 case preoperative bronchoscopy revealed an area of ulceration around the left anteromedial basal-segment bronchus. Biopsy of this was reported grade 4 squamous-cell carcinoma. Total left pneumonectomy and hilar-gland dissection were carried out. The primary tumor was relatively small, 2.5 by 2.0 by 2.0 cm. There were no evident lymphatic metastases, and the main bronchus had been resected 2.5 cm. proximal to the tumor. Six months postoperatively persistent cough and hemoptysis developed. Two months later (8 months following pneumonectomy) hoarseness was noted and a broncho-

Table IV. Summary of Data for Cases in Group 4

CASE	PREOPERATIVE BRONCHOSCOPY	OPERATION	CONSIDERED PALLIATIVE?*	LYMPH-NODE INVOLVEMENT	MARGIN OF RESECTION (CM.)	INTERVAL: RESECTION TO RECURRENCE (MONTHS)	RECURRENCE DIAGNOSED BY:	INTERVAL: RESECTION TO DEATH (MONTHS)
15	Questionable infiltration left main bronchus Biopsy negative	Left pneumonectomy	No	Yes	2.75	6	Cells in sputum	Unknown
16	Lesion left main bronchus Biopsy positive	Left pneumonectomy	No	No	2.5	6	Cells in sputum and biopsy	12
17	Tumor level of middle lobe bronchus Biopsy positive	Right pneumonectomy	Yes	Yes	2.5	6	Biopsy	Unknown

*Operation considered palliative at conclusion of resection.

Table V. Summary of Data for the Single Case in Group 5

CASE	PREOPERATIVE BRONCHOSCOPY	OPERATION	CONSIDERED PALLIATIVE?*	LYMPH-NODE INVOLVEMENT	MARGIN OF RESECTION	INTERVAL: RESECTION TO RECURRENCE (MONTHS)	RECURRENCE DIAGNOSED BY:	INTERVAL: RESECTION TO DEATH (MONTHS)
18	No lesion seen No biopsy	Right, middle, and lower lobectomy	No	No	No tumor in major bronchi	14	Bronchial smear	24

*Operation considered palliative at conclusion of resection.

scopic examination substantiated the diagnosis of vocal cord paralysis and recurrent carcinoma in the bronchial stump. Death occurred in this case 12 months postoperatively. Histologically, there was marked epithelial metaplasia in the proximal tumor-free resected bronchus in this and the other 2 cases in this group. The possible significance of this finding will be discussed in a later section.

Group 5.—In this category (Table V), there is 1 case which represents the only peripheral tumor which, to our knowledge, has recurred in the bronchial stump. The patient was the only woman in the series. Preoperative bronchoscopy failed to reveal any evidence of tumor but sputum contained malignant cells. Because the patient was 72 years old and there was no apparent lymphatic involvement, resection of the right middle and lower lobes was carried out. The tumor, 2.5 cm. in diameter, was located in the depths of the middle lobe. No involved lymph nodes were found, and serial sections of the proximal middle and lower lobe bronchi contained no evidence of tumor or epithelial metaplasia.

Hemoptysis developed 14 months postoperatively, at which time bronchoscopy revealed a firm, ragged lesion on the anteromedial wall of the right main bronchus about 2 cm. from the carina. Smears from this lesion contained malignant cells. Death occurred 2 years following pulmonary resection.

RÉSUMÉ OF FINDINGS AND COMMENT

There are no available reports concerning the recurrence of carcinoma in the bronchial stump. The true incidence of this entity is impossible to determine from a study of purely surgical material. Aufses states that he has definite evidence of stump recurrence in 6 cases of the 165 pulmonary resections for carcinoma performed between 1935 and 1948 at the Mount Sinai Hospital, New York. The present report is based on a study of 18 patients who developed recurrence in the bronchial stump after pulmonary resection for carcinoma at the Mayo Clinic. These cases were taken from a total of 631 patients who underwent resection for bronchogenic carcinoma prior to September, 1952.

All except 1 of the patients in this series were men, and the average age of the patients was 55.3 years. The average duration of symptoms prior to resection was 7.6 months.

Preoperative cytologic examination of sputum, bronchial washings, bronchial secretions, or more than one of these was carried out in 14 of the cases and gave positive results in 11.

Bronchoscopic examination was performed prior to thoracotomy in all 18 of these cases. In 11 cases a tumor was seen in either the right or the left main bronchus, and in 10 of 15 cases in which biopsy was performed the result was positive. In 3 cases some narrowing or infiltration was noted in or around the orifice of a secondary bronchus but biopsies were reported negative. In 4 cases the lesion was not visible bronchoscopically. The region of the carina appeared grossly normal in all 18 instances.

The findings at preoperative bronchoscopic examination are of special interest in view of the recent work of Rabin and his associates with biopsy near the carina. These workers stated that they have found the procedure extremely useful, since it produced positive findings in close to 20 per cent of patients who were considered otherwise operable. Apparently the result of biopsy may be positive even in the presence of grossly normal mucosa.

So-called carinal or paracarinal biopsies were not done in the cases in this series. Had this procedure been carried out, it is conceivable that results would have been positive in at least a few cases.

All the tumors which, in the Mayo Clinic series, have recurred in the bronchial stump were squamous-cell carcinomas of high histologic grade (Broders). The size varied from 1.5 cm. to 6.0 cm. in diameter. Lymph nodes in the hilus were involved in 12 cases.

In 16 cases a main bronchus was involved by the primary tumor at the time of pneumonectomy. These were equally divided between the right and left sides. The two tumors not originally involving a main bronchus were located in the right lower and in a peripheral right middle lobe bronchus, respectively. They were each treated by resection of the right middle and lower lobes with preservation of the upper lobe.

Symptoms of bronchial-stump recurrence appeared at intervals varying from 5 weeks to 24 months following resection. Attempts to explain many of these recurrences must, of necessity, be somewhat speculative. This is not true of the 5 cases in which gross or microscopic carcinoma was found at the line of bronchial resection. Obviously the "recurrence" was due to residual tumor in these cases. The situation, however, wherein resection of a relatively small, peripherally located carcinoma without demonstrable lymphatic spread is followed in 14 months by a bronchial-stump lesion identical histologically to the primary growth is more difficult to explain. It seems probable that by existing standards the initial resection was adequate. The possibility of an occult metastasis or seeding to the region of the main bronchus from the primary tumor at the time of operation must be considered. Similarly, a second primary carcinoma is a definite possibility. Spain and Parsonnet have reported the case of a 92-year-old woman whose bronchial tree showed multiple primary neoplastic changes. Williams has presented the case of a patient who had two separate invasive bronchogenic carcinomas.

The problem of in situ carcinoma is pertinent. Papanicolaou and Koprowska have reported a case of epidermoid carcinoma in situ of a right lower lobe bronchus. The tumor was detected by cytologic examination of sputum and its presence was proved at necropsy in one of a number of random blocks from the right lower bronchial tree. Umiker and Storey, Black and Ackerman, and Carlisle, McDonald, and Harrington have also demonstrated in situ changes in bronchial mucosa.

Minute parenchymal tumors believed to represent early bronchogenic carcinoma have been reported by Petersen, Hunter, and Sneeden. Aufses and Neuhof have reported 2 cases of very small bronchial cancer. Wierman has recently presented a study of 5 cases of occult bronchogenic squamous cell carcinoma. All these tumors involved major bronchi and were not readily apparent. One of the carcinomas was in situ and the other four were grossly minute, invasive lesions with in situ changes in the surrounding epithelium.

The question of what constitutes an adequate margin of bronchial resection is not easy to answer. The work of Griess, McDonald, and Clagett indicates that in squamous cell bronchogenic cancers the minimal safe distance for excision of the bronchus is 1.5 cm. from the proximal limits of the tumor. By use of formalin-preserved surgical specimens, microscopic extension of some squamous cell cancers was demonstrated in the submucosal bronchial layers for distances varying from 0.3 cm. to 1.5 cm.

Obviously a certain amount of shrinkage occurs in all tissues preserved in formalin solutions. It is not possible to state exactly how important this factor has been in the present study, but shrinkage of a semirigid, cartilage supported bronchus is certainly much less than the high degree of shortening which takes place in the preservation of segments of alimentary tract and similar tissues.

It has been noted that epithelial metaplasia was present in the resected bronchi proximal to tumor in many cases in this series. The epithelial changes varied in degree from case to case and within the individual specimens. The earliest change noted was disorganization and palisading of cells in the basilar layers of otherwise normal bronchial mucous membrane. Other changes included pleomorphism, frank squamatization, and cellular mitoses. Several examples of epithelial metaplasia are illustrated in Figs. 1, 2*b*, and 4.

The difficulty in distinguishing between so-called metaplastic and neoplastic changes in tissues has been noted by several authors. Williams' case has already been mentioned. There were extensive intra-epithelial carcinomatous changes in the bronchial epithelium. Williams stated that the epithelial changes in this case might be considered by some pathologists to be so-called carcinoma in situ while others would regard them as cellular metaplasia. Spain and Parsonnet have also emphasized the difficulty in differentiating between cellular metaplasia and neoplastic changes.

Carlisle, McDonald, and Harrington, in a study of bronchogenic squamous cell carcinomas at the Mayo Clinic, noted that a small zone of mucous membrane adjacent to many of these tumors contained in situ changes. Similarly, Womack and Graham have noted a case of bronchogenic carcinoma in which the mucosal epithelium adjacent to the tumor showed patchy metaplasia with considerable mitotic activity. They considered this metaplasia to be a precancerous condition. Reingold and associates, in a necropsy study of 60 cases in which the patients had died of bronchogenic carcinoma, reported several instances in which the gradation from normal bronchial mucosa through squamous metaplasia to epidermoid carcinoma was observed.

According to Willis, Karl Tiersch, in 1865, suggested that the recurrence of an epithelial tumor following surgical excision was not always due to incomplete removal of tumor tissue, but was sometimes due to new carcinomatous change in the predisposed epithelium of the region.

It is rather disturbing that the interval from pneumonectomy to recurrence was nearly the same in cases with carcinoma present at the line of bronchial resection as in some cases with only metaplastic epithelium in the proximal excised bronchus. It is possible that the latter, morphologically still benign epithelium, may in reality be carcinoma. Perhaps the same amount of time is necessary for residual carcinoma in the bronchial stump to reassert itself as is required for metaplastic epithelium in the carinal region to bleed and initiate coughing. Further study of this problem seems necessary. It may be that in a case of bronchogenic squamous-cell carcinoma the finding of metaplastic epithelium near the carina is of nearly as much prognostic significance as is the presence of frank carcinoma in this region.

Blood-vessel involvement was demonstrated in a bronchus in only 1 case in this series. In this case, blood vessels in the bronchial wall were involved at a point 1.0 cm. from the proximal resected end of the main bronchus. This finding could certainly account for the bronchial-stump recurrence. It is of interest, though, that Ballantyne found blood-vessel involvement in 80 per cent of bronchogenic squamous-cell carcinomas. He noted, however, that blood vessels were involved in 16 of 27 patients who survived 2 to 5 years after pulmonary resection for squamous-cell cancer and concluded that the presence of malignant cells in a blood vessel does not make metastasis a certainty.

As might be expected, the commonest symptom of bronchial-stump recurrence was cough, which occurred in some degree in all cases. This was usually productive and in 16 cases hemoptysis appeared. Hoarseness due to paralysis of the left vocal cord occurred in 4 cases. Cough and hemoptysis following pulmonary resection for carcinoma may be due to benign granulation tissue. Persistence of these symptoms must be viewed with suspicion, however, and bronchial-stump recurrence should be considered.

Bronchial-stump recurrence appears to be of serious prognostic significance. Of 13 cases in which data are available, only 4 patients survived 2 years or more following resection despite treatment in several instances with deep x-rays or locally implanted radon seeds or both.

SUMMARY AND CONCLUSIONS

1. A clinicopathologic study has been presented of 18 patients in whom bronchial-stump recurrence developed following pulmonary resection for a squamous-cell carcinoma of the lung.

2. In 16 cases a main bronchus was involved by the primary tumor. Eight of these were on the right and the same number were located in the left lung. In 12 cases, lymph nodes in the pulmonary hilus were metastatically involved.

3. In 11 cases, the tumor was visualized bronchoscopically prior to resection. The region of the carina was not grossly involved in these patients preoperatively. On the basis of the present study, however, it is believed that bronchoscopic biopsy near the carina might have been helpful in assessing operability in several instances.

4 In 5 of the 18 cases in this series, pathologic examination of the surgical specimens revealed the presence of carcinoma at the proximal end of the resected bronchus In 9 cases the margin of resection beyond the tumor was less than 1 5 cm, and the bronchial mucosa proximal to tumor was metaplastic in 7 of these In 3 of the 18 cases, the bronchus was resected at least 2 5 cm beyond the tumor The proximal bronchial epithelium was metaplastic in all 3, however The tumor in 1 case involved no major bronchi and the bronchial epithelium showed no evidence of metaplasia

5 Metaplasia of the bronchial epithelium was a common finding in this study, and its possible importance in the etiology of certain bronchial stump recurrences has been discussed

6 In this study the interval from pulmonary resection to the appearance of symptoms of bronchial stump involvement varied from a few weeks to almost 2 years

7 The most constant symptom of bronchial stump recurrence was cough, which occurred in some degree in all cases Hemoptysis appeared in all but 2 cases In 4 cases hoarseness developed as a result of vocal cord paralysis The occurrence and persistence of cough and hemoptysis after resection for bronchogenic carcinoma must be considered a recurrence in the bronchial stump until proved otherwise

8 The diagnosis of bronchial stump recurrence was established by bronchoscopy in 16 cases and by cytologic examination of sputum in the remaining 2 cases in this series

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ABDOMINAL PAIN FOLLOWING SURGICAL CORRECTION OF COARCTATION OF THE AORTA: A SYNDROME

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OUR attention was recently focused on singular events in the postoperative course of 2 patients on the cardiovascular service of this hospital. These men had had surgical excision and repair of typical thoracic aortic coarctations performed within a few days of each other. Thereafter, both, in the early postoperative period, developed profound abdominal pain and tenderness with marked leukocytosis and fever for which we had no ready explanation. Within ten days these findings subsided with conservative therapy. Our associates volunteered similar experiences and this induced us to review the postoperative course of patients treated here for this anomaly.

Sealy,[1] in 1953, reported a case of abdominal distress with development of intestinal infarction following repair of aortic coarctation and Lober and Lillehei[2], from this Clinic, wrote of two instances of necrotizing panarteritis of the abdominal viscera following resection of coarctation. Perez-Alvarez and Oudkerk[3] also reported an instance of similar nature.

Since 1950, the diagnosis of coarctation has been coded in this hospital 88 times. Seventy-three of these patients came to surgery and of this group 64 survived at least the early postoperative period. Eighteen patients in this group had significant abdominal findings and symptoms in the early postoperative period. We have arbitrarily divided these 18 patients into three groups on the basis of intensity of signs and symptoms.

The first group, which showed the mildest symptoms, consisted of 10 patients (one female) ranging in age from 2½ to 34 years (average 23 years). All of these complained of generalized abdominal pain during the first to third postoperative days. One or more of the following problems was present in each instance: abdominal distention, gastric dilatation, abdominal tenderness, ileus, or vomiting. Leukocytosis was present (13,000 to 20,000) with mild temperature elevation (101° F.). These complications responded to restriction of oral intake, gastric suction, and intravenous administration of fluids. Since such problems may arise following thoracotomy, for whatever reason, we cite this group only to point out the incidence of abdominal distress following resection of coarctation. The following case is an example from this group:

D. J. (UH #857949).—The patient was a 28-year-old white man with a history of "dizzy spells" for several years and dyspnea on exertion of six weeks' duration. Physical

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findings revealed a 144 pound individual with a right brachial artery pressure of 150/90 and left pressure of 150/88 There was a loud precordial systolic murmur that radiated to the back Femoral pulses were not palpable

X ray showed slight left ventricular enlargement and the electrocardiogram indicated left axis deviation

Resection of the coarctation was done on May 21, 1953 The proximal aorta was 3 cm in diameter with a 4 mm opening in the coarctate segment The ductus arteriosus was not patent

On the third postoperative day, the patient developed generalized abdominal pain, distention, and diarrhea The WBC was 17 100, and the temperature was 101° F The patient required 50 to 75 mg of Demerol for six days The symptoms relented after three days of gastric suction and intravenous fluids

The blood pressure on the third postoperative day was 140/90 The femoral pulses were of good quality At six month check, the blood pressure was 126/86

A second group of 4 patients (all male) experienced similar difficulties but of more serious nature The ages ranged from 4 to 36 years (average 19 5), the onset of symptoms was in the first postoperative week and persisted for seven to ten days Here again abdominal distress was significant and x ray examination showed that ileus was largely confined to the small bowel Severe generalized abdominal pain and tenderness with rebound were manifest Vomiting occurred on several occasions Pyrexia (103° F) and leukocytosis (15,000 to 30,000) were consistently present without obvious explanation

These patients were studied during and subsequent to the acute episode from which they all recovered by conservative means of treatment The only abnormal laboratory finding was the elevation of the white blood count Serum amylase studies were normal Muscle biopsy of the lower extremity in one instance was not revealing X-ray studies of the intestinal tract during subsidence of the acute episode presented no abnormality in one instance The following case history is an example from this group

R V (UH #878584) —The patient was a 22 year old man who gave a vague history of dizzy spells His weight was 159 pounds and blood pressure on admission measured 210/108 A systolic precordial murmur was heard The femoral pulses could not be felt

X ray showed left ventricular enlargement and the electrocardiogram demonstrated left ventricular hypertrophy

Surgical correction of the coarctation was performed on Jan 6, 1955 Arterial collateral channels were extremely rich in the chest wall The aorta measured 1 cm in diameter with a 1 mm opening in the coarctate segment

On the tenth postoperative day, the patient developed severe abdominal pain with distention and vomiting The temperature was 101° F The WBC was 29,000 with 88 per cent polymorphonuclear neutrophil leukocytes The patient described a "pumping" pain in the legs With gastric suction and intravenous fluids, the patient slowly improved He required 5 to 10 mg of methadon for pain for ten days after this acute episode for which no explanation could be found The blood pressure in the right arm measured 130/80 at the time of its onset Since discharge from the hospital the patient has done well

The third group of 4 patients (4 males, aged 3 to 46 years) were subjected to laparotomy within two weeks following correction of the coarctation The diagnosis of appendicitis was entertained in 3 of these patients The striking feature of this group is the similarity of abdominal findings Poorly localized abdominal pain and tenderness beginning on the seventh to tenth postoperative day with leukocytosis and fever, were associated with nausea and vomiting with rebound tenderness. This led to the diagnosis of a "surgical abdomen," but in no instance was a specific diagnosis made preoperatively.

Two of these cases were previously reported by Lober and Lillehei.[2] Here, necrotizing panarteritis indistinguishable from periarteritis nodosa was found with infarction of portions of the small intestine. One patient succumbed from a ruptured splenic aneurysm. The second, subjected to a bowel resection by one of us (F. J. L.), recovered. A third patient had a negative laparotomy but expired one month later from a ruptured dissecting aneurysm of the thoracic aorta. The fourth patient in the group had only enlarged mesenteric lymph nodes found at surgery and he subsequently recovered. The following 2 cases are examples from this group:

J. S. (UH #850534).—The patient was a 3-year-old boy who was admitted to the hospital with a history of episodes of cyanosis and shortness of breath on exertion and congestive heart failure.

Physical examination revealed a blood pressure of 134/60 right arm and 130/60 left arm. A precordial systolic murmur was heard which radiated to the back. The femoral pulses were absent.

X-ray showed right and left ventricular enlargement and the electrocardiogram showed right axis deviation.

Surgery was performed on March 3, 1953, and minimal arterial collaterals were encountered in the chest wall. The ductus arteriosus was closed. The aorta measured 1 cm. in diameter and the coarctate segment was 2 mm. in diameter. Arteriosclerotic changes were noted on microscopic section of the aorta immediately adjacent to the constricted area.

On the seventh postoperative day the child developed abdominal pain, rigidity, and generalized tenderness. The WBC was 16,200. The rectal temperature was 101° F. Two days later the symptoms seemed progressive and laparotomy was done with findings of mesenteric adenitis. The patient recovered uneventfully and was discharged on the eighteenth day following the original surgery.

E. H. (Associated Hospital).—This 46-year-old white man had been treated for pulmonary tuberculosis in February, 1953, by right upper lobectomy. He had no symptoms directly related to the coarctation.

Physical examination showed blood pressure in the upper extremities of 200/100 with absent femoral pulses. An apical systolic murmur was heard.

X-ray examination showed left ventricular enlargement.

Corrective surgery was done on May 6, 1953. A typical coarctation was resected with end-to-end anastomosis.

On the eighth day after surgery, the patient developed fever and vomiting. Two days later the abdomen was distended, tender, and rigid. The temperature at this time was 103° F. Abdominal exploration was carried out, and free pus was found in the peritoneal cavity. Gangrenous patches were present along the ileum and distal part of the jejunum. Thirty-eight centimeters of the small intestine were resected. The mesenteric vessels were not grossly abnormal. The postoperative course was not remarkable.

The pathologic changes of medial necrosis with cellular infiltration and fibrinoid deposits, similar to periarteritis nodosa, have been described elsewhere.[2]

Table I sets forth the general data regarding this group of patients with coarctation of the aorta. In this series of patients receiving surgery, 9 expired on the table and this number, in conjunction with those who did not receive surgery, left a total of 64 patients who survived sufficiently long into the postoperative period to be evaluated. The 18 cases with significant abdominal findings constitute 28 per cent of this group. The total number of women in the surgical group was 25 (28.2 per cent).

TABLE I COARCTATION OF THE AORTA
(73 PATIENTS OPERATED UPON)

	AGE (YEARS)				
	<1	1 5	6 25	>25	TOTAL
Number of Patients	15	8	38	12	73
Deaths	8	0	3	2	13
Abdominal Findings—					
Moderate I	0	2	4	4	10
Severe II	0	1	2	1	4
Surgical III	0	1	2	1	4

An analysis of the 18 patients under consideration reveals some interesting points The average age of Group I was 23 years, Group 2 was 19 5 years, and Group 3 was 19 years, while the average over all age was 11 years There was one woman (5 per cent) in the group of 18 patients with abdominal symptoms, compared to the over all figure of 28 2 per cent The other 2 cases reported in the literature were also men An effort was made to correlate the pre and postoperative (seventh to tenth day) blood pressures of the group of 18 with those of the whole group of surgical patients and generally the patients developing abdominal complications seemed to have an earlier and more marked drop in pressure, especially those in the older age group (Table II) No consistent trend could be detected in comparison with preoperative palpability of femoral pulse or estimated degree of chest wall collaterals at the time of surgery

Pentothal sodium anesthesia was routinely employed The *postoperative* care of all surgical cases was essentially the same Penicillin and streptomycin were employed for five to seven days and in only one instance was there overt evidence of sensitivity reaction to these agents

DISCUSSION

Since necrotizing arteritis was found in tissue removed at surgery from 2 of our patients (previously reported), and in the case cited by Perez Alvarez,[3] the possible relationship of this process to periarteritis nodosa has been considered Inasmuch as the value of study of the rare entity frequently lies in the elucidation of the common entity, this pursuit seems to have merit

Since the original description in 1866 by Kussmaul and Maier, periarteritis has been alternately considered a distinctly separate disease and a general term for a group of disorders *based pathologically* on necrotizing inflammation of muscular arteries Zeek and others[4,5] have helped to resolve this problem by listing periarteritis nodosa as one distinct form of necrotizing angitis characterized histologically by inflammation and fibrinoid necrosis of the muscular arteries That hypersensitivity reactions are its sole etiology seem no longer tenable A hypertensive factor in the etiology of periarteritis nodosa seems quite appealing in view of the laboratory demonstrations by Kempner and co workers,[6] of the development of this disorder in rats, secondary to elevations of arterial pressure The liver arterialization studies of Rather and Cohn[7] in pro necrotizing lesions in the hepatic vascular tree and the evidence that inc intravascular tension is directly concerned in the production of acute a necrosis (Byrom and Dodson[8]) add weight to the thesis that *intrava* trauma may produce definite acute pathologic changes in the *arterial wal*

TABLE II. AVERAGE BRACHIAL ARTERIAL BLOOD PRESSURES
(BEFORE AND AFTER CORRECTION OF COARCTATION)

	ENTIRE SURGICAL GROUP								PATIENTS WITH ABDOMINAL FINDINGS					
	<1		1-5		5-25		>25		1-5		5-25		>25	
AGE (YEARS)	BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER
	128/96	109/50	154/68	126/x	155/96	139/82	176/113	164/108	133/70	120/60	178/101	141/85	196/100	146/90
Systolic drop		19		28		14		12		13		37		50
Diastolic drop		46		x		14		5		10		16		10

The confinement of periarteritis lesions to the vessels subject to systemic blood pressure and the freedom of the pulmonary system (except in instances of pulmonary hypertension) from vascular necrosis suggests that some limited factor is at play in this disease

The one common factor in all cases studied was the sudden increase in pulsatile pressures in the arterial system distal to the coarctation The signs and symptoms in our cases were confined to the visceral areas subject to this increased pressure head This appeared especially true of the structures supplied by the superior mesenteric artery

The abdominal pain manifest in all of our patients may be related to the arterial distention stimulating viscerosensory fibers in the vessel wall It is noted that one of the patients complained of epigastric pain, "pumping" with each heartbeat The prolonged requirement for narcotics (five to ten days) in our series of postoperative cases was consistently noted

That the majority of the postoperative patients with coarctation can tolerate this change must represent an adaptation less well handled by the 18 cases in point It would seem that the patients demonstrating significant abdominal findings represent a varying pathologic response to the elevated arterial pressures in the postcoarctation vasculature ranging from temporary functional disturbance to local lysis of overstretched medial muscular fibers, vascular occlusion, and tissue death The two instances in this series of pathologic changes identical to periarteritis nodosa were limited to vessels beyond the coarctate area and represent an extreme expression of the devastating influence of acute overdistention of the arterial tree*

We have no ready explanation for the preponderance of males in this group showing abdominal distress after corrective surgery The tendency for the older patient to develop the syndrome may in part be due to the inability of the older vessels to adapt to the elevated pressures That the age of the vessel is not gauged wholly by the chronological age of the patient is indicated by 2 cases of youthful patients in the group of 18 (ages, 2½ and 5) in which arteriosclerotic stages were noted in the aorta proximal to the coarctation

SUMMARY

A series of 18 cases presenting similar signs and symptoms of varying intensity referable to the abdomen, following repair of coarctation of the aorta, is reported Possible etiological factors are discussed We feel that this entity justifies the term "syndrome" Cardiovascular surgeons should be alert for this complication Certainly this problem deserves further study

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*These cases may be examples of periarteritis nodosa in a limited portion of systemic circulation

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CHONDROSARCOMAS OF THE THORACIC SPINE

Report of Two Cases

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As the scope of thoracic surgery has increased in recent years, it has not only enabled more accurate diagnosis but also has stimulated greater interest in lesions which were formerly relegated to the field of academic interest only. Chondrosarcoma of the thoracic spine falls in this category.

Presented herewith are 2 case reports of thoracic vertebral chondrosarcomas which present rather unique features both clinically and pathologically.

The rarity of chondromatous tumors of the thoracic spine is attested by the sparse literature on the subject. Geschickter and Copeland[1] in reporting 321 instances of osteochondromas from the Johns Hopkins Hospital up to 1949 found only one lesion of the thoracic spine. Weisel, and Ross[2] reported a chondrosarcoma in 1950 and Ilgenfritz[3] reported a most unusual osteochondroma of the upper spine in 1951. We have been unable to find previous reference or record of resection of chondrosarcomas of the thoracic spine

CASE REPORTS

Case 1.—A white man, aged 32 years, was referred for thoracotomy because of a homogeneous ovoid opacity in the posterior mediastinum adjacent to the right lung field and at the level of the sixth thoracic vertebra This had been asymptomatic and was first discovered on a routine Army chest x ray examination. The lesion measured about 5 cm in maximum diameter. Past history was noncontributory. Bronchoscopy was unremarkable

Exploratory thoracotomy demonstrated a smooth walled mass about 8 0 by 6 0 cm. in diameter which was brittle to touch It was attached to the body of the sixth thoracic vertebra. The mass was excised, and the vertebral body curetted.

Grossly, the lesion was a mass of hemorrhagic and soft, translucent, mucoid material with calcific particles scattered throughout. Histologic sections (Figs. 1 and 2) demonstrated hyaline cartilage, bone, and myxomatous tissue. The diagnosis of chondrosarcoma was arrived at mainly because of plumpness and hyperchromatism of the chondrocytic nuclei. The myxomatous portions exhibited nuclear hyperchromatism and irregularity in addition to necrosis with hemorrhage. These were presumed to represent degenerative changes.

The patient made an excellent recovery. A two year follow up finds him well and active with no recurrence.

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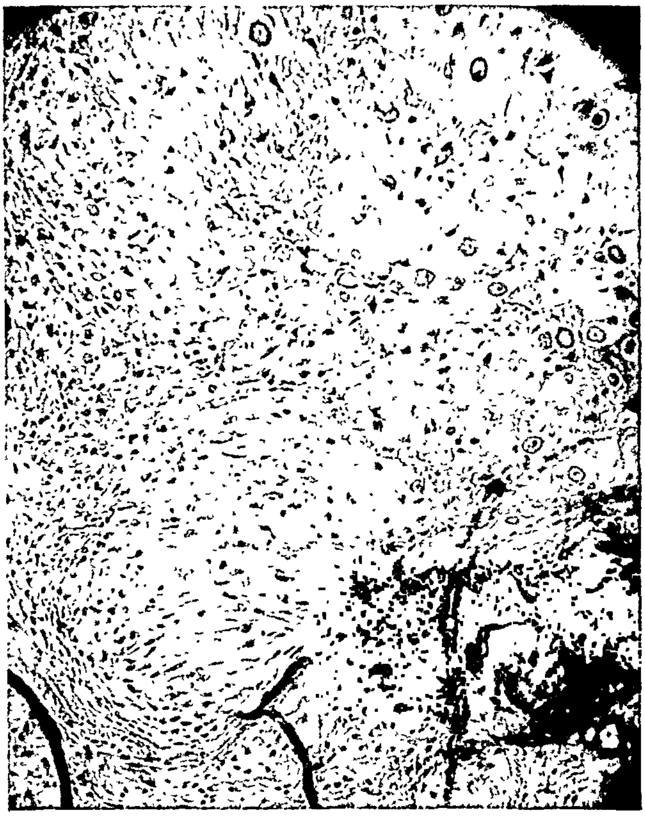

Fig. 1.—Case 1. Histologic section.

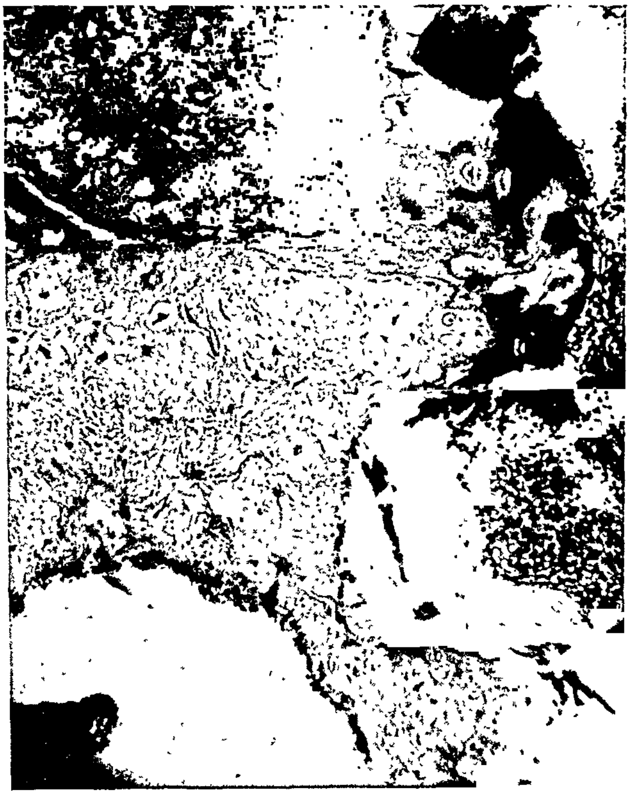

Fig. 2.—Case 1. Histologic section.

CASE 2—A white woman, aged 20 years, had been in good health until she was hospitalized for right lateral thoracic pain of pleuritic character Chest x rays disclosed a right pleural effusion (Fig 3) Thoracentesis demonstrated bloody serous fluid Cytologic studies of this were negative Postthoracentesis x rays (Fig 4) showed a partly calcified rounded opacity of the posterior mediastinum arising at the level of the seventh thoracic vertebra and extending into the right lung field This measured about 10 0 cm in maximum diameter

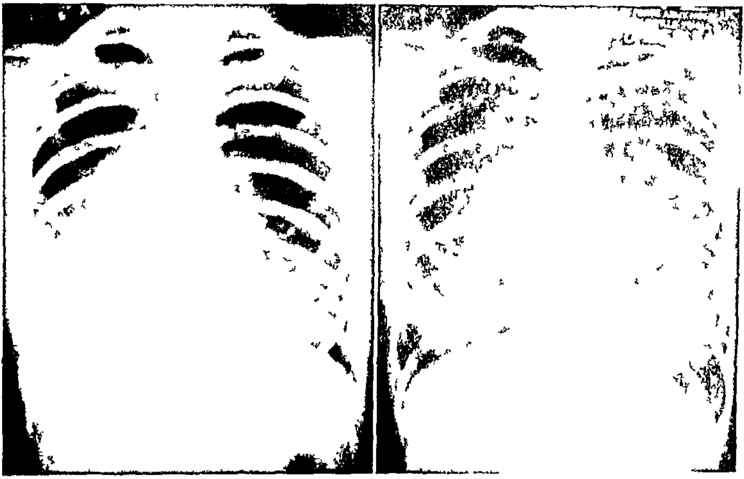
Fig 3 Fig 4

Fig 3—Case 2 Before aspiration of hemothorax
Fig 4—Case 2 After aspiration of hemothorax

Exploratory thoracotomy presented an ovoid mass, the upper two thirds of which was covered by a smooth surface, presumably parietal pleura The lower portion was fragmented hemorrhagic, and focally myxomatous A fresh clot of blood extended downward from the lesion The mass was rather tenuously attached to the body of the seventh thoracic vertebra The lesion was removed, and the area of attachment curetted A nearby mediastinal lymph node measuring 2 0 cm in maximum diameter was removed separately Histologic sections demonstrated a lesion essentially similar to that of Case 1 (Fig 5) A particularly interesting finding was the presence of neoplastic tissue in a lymphatic vessel adjacent to the lymph node (Fig 6)

The patient recovered uneventfully from surgery (Fig 7) She is now well with no evidence of recurrence six months following surgery

COMMENT

Our experience with these 2 patients has called to our attention an entity which is not considered often in the diagnosis of posterior mediastinal lesions The radiologic *findings were sufficiently distinctive as to suggest at least a* correct diagnosis of chondrosarcoma in these cases In our second patient, the appropriate diagnosis was considered prior t[illegible] alth[illegible] ne[illegible]fibroma was somewhat more str[illegible] suggested

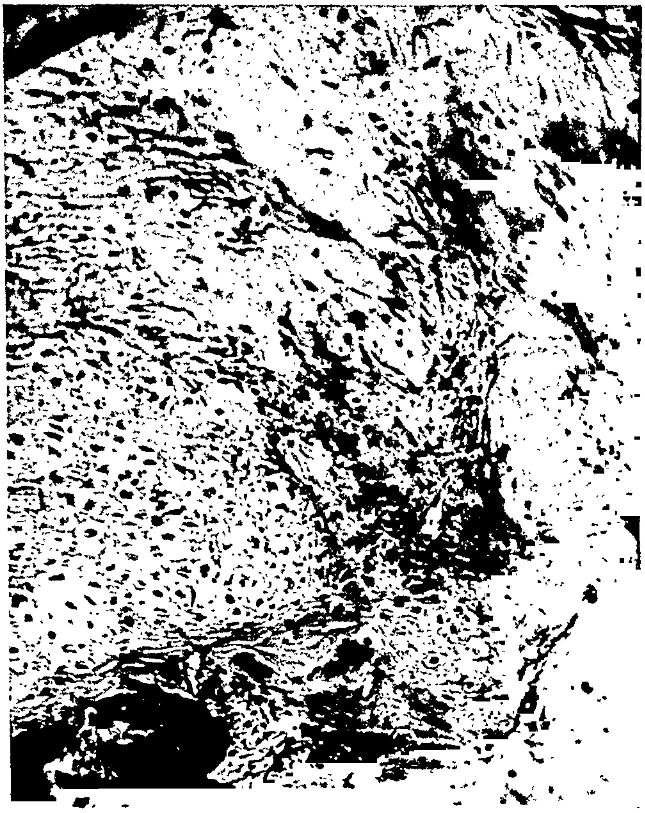

Fig. 5.—Case 2. Histologic section.

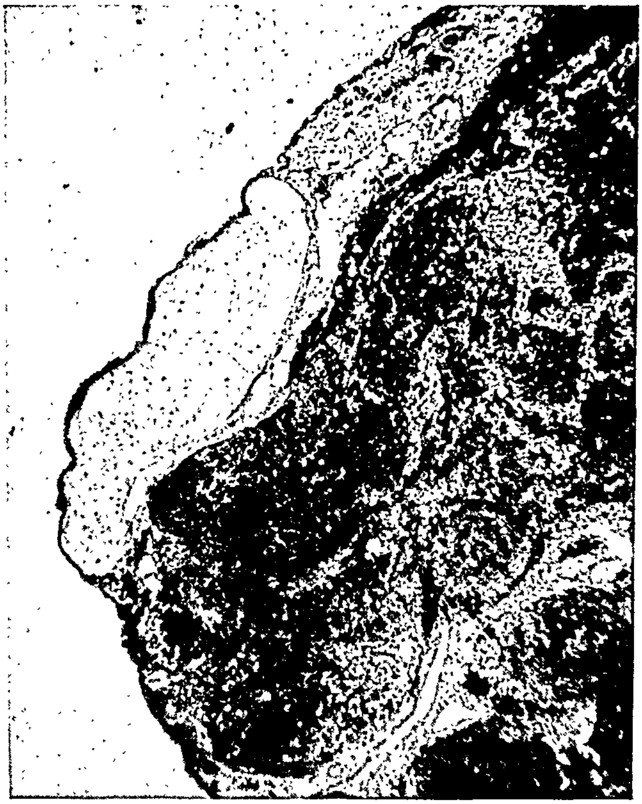

Fig. 6.—Case 2. Histologic section.

The gross findings in each of these patients were unique in our experience, namely, the presence of a brittle mass composed largely of soft, gray, translucent mucoid material. In retrospect, these gross findings at surgery should have suggested the diagnosis of myxoma or chondrosarcoma in each instance.

Since chondrosarcomas are a rarity and since myxomatous changes are characteristic of chondrosarcoma, this point is considered worthy of emphasis. This is particularly important since such tissue does not lend itself to frozen section diagnosis.

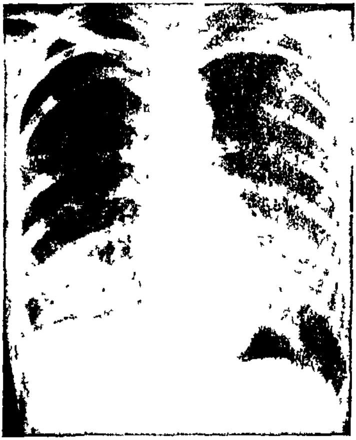

Fig. 7.—Case 2. Postoperative x-ray.

A particularly interesting finding in the second case was the lymph node metastasis. Lymphogenous metastases are rarely reported secondary to chondrosarcoma. The metastatic involvement in this case offers corroborative biologic evidence of the malignant nature of the lesion. This is important inasmuch as the histologic criteria of malignancy in the cases we are reporting, as well as in many of the cases of chondrosarcoma reported elsewhere, are of rather equivocal character. Mitoses are rarely seen in any significant number in chondrosarcomas inasmuch as growth of cartilage is by amitotic division. We saw no mitoses in the tissue from our cases. On the other hand, the criteria of malignancy as set forth by Lichtenstein and Jaffe[4] were satisfied. These include plumpness of nuclei, more than occasional cells with double plump nuclei and giant cartilage cells.

It is not possible to indicate with certainty the histogenesis of the lesions in these 2 cases. Various authors have suggested that chondrosarcomas arise either from cartilagenous rests or from osteochondromas. O'Neal and Ackerman have mentioned that it is frequently difficult to determine the manner of origin of malignancies of cartilagenous nature. Although both bone and well-differentiated cartilage were found in each of these lesions, it was not oriented in such a manner as to suggest origin from osteochondromas. In other words, it was not possible to delineate well-differentiated bone overlaid by a cartilaginous "cap." It would seem worth while to attempt to prove such a relationship since this would provide additional information relative to the proper handling of osteochondromas, although most authors agree that the incidence of malignancy in osteochondromas is very low.

It seems worth mentioning that the location of these lesions adds some support to the concept of Roberg[6] that cartilaginous neoplasms situated near the body axis are more apt to be malignant.

SUMMARY

Two unusual cases presenting chondrosarcomas of the thoracic vertebrae are reported. Diagnostic and pathologic implications have been discussed.

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CONGENITAL EVENTRATION OF THE DIAPHRAGM

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CONGENITAL eventration of the diaphragm is an unusual condition. The normal diaphragmatic muscle is replaced by a thin, translucent, fibrous membrane. Usually only the central dome is affected, causing the diaphragm to balloon high up into the pleural cavity. Rarely is the entire diaphragm involved.

Although over 300 cases[1] have been reported in the literature, successful surgical repair in infancy is rare. Arnheim[2] in a review of the literature, in 1954, collected a series of 8 cases. In 1954, Laxdal and associates[1] reported an additional 2 cases. One infant, aged 7 weeks, was operated upon successfully; the other patient, aged 28 days, died without surgery.

In 1952, Ravitch and Handlesman[3] reported surgical repair of small defects in the right diaphragm in a series of 5 infants. These were small areas of eventration with herniation of the liver. These cases cannot be classified as true eventration of the diaphragm.

In 1947, Bisgard[4] performed the first successful surgical repair of eventration of the diaphragm in infancy.

The case reported here is the tenth infant to undergo successful surgical repair, and the fourth case with involvement of the right diaphragm.

A different surgical technique is demonstrated which preserves the normal function of the diaphragm.

CASE REPORT

T. S., a white male infant, was delivered normally at St. Francis Hospital on April 12, 1953. The birth weight was 8 pounds, 5 ounces. He was re-admitted to the hospital on May 14, 1953, on the service of Dr. Edward Giunta.

The mother stated that the baby had a chronic "brassy" cough, noted one week after birth. The baby did not take his feedings well and had failed to gain weight. He had occasional periods of dyspnea and slight cyanosis, especially during feedings.

Physical examination revealed a moderately undernourished 4-week-old infant, weighing 8 pounds, 7 ounces. There was slight cyanosis of the lips. On inspiration, there was reduced expansion of the right hemithorax. Breath sounds were decreased on auscultation over the right base of the chest.

Roentgenologic examination of the chest revealed an abnormally high position of the right diaphragm. The dome of the diaphragm extended to the third anterior intercostal space (Fig. 1). Fluoroscopy of the chest revealed decreased excursion of the right diaphragm. Roentgenologic examination of the esophagus and stomach revealed normal position and contour of the esophagus. The fundus of the stomach was in normal position,

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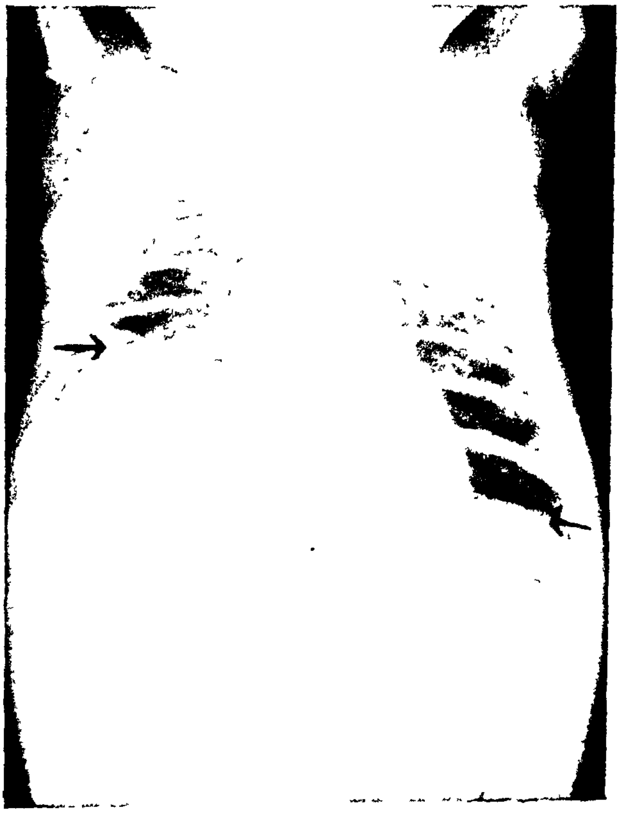
but the pylorus and first part of the duodenum were high and to the right of the midline. The stomach was slow to empty. The remaining laboratory work was within normal limits.

Because of the severity of the symptoms, and failure to gain weight, surgical correction of the diaphragmatic eventration was decided upon.

Operative Procedure.—The baby was operated upon by one of us (R. A. D.) on May 25, 1953. Endotracheal anesthesia, using cyclopropane, was administered by Dr. W. O. McQuiston. A cut-down was performed on an ankle vein for administration of fluid and blood.

The chest was opened through the right eighth interspace. The dome of the diaphragm was found to consist of a thin, translucent, fibrous membrane, and extended nearly to the apex of the pleural cavity. It was much higher than appeared on the chest roentgenogram. The liver could be seen underneath the thin membrane. The right lower lobe showed small areas of atelectasis, due to external compression. The periphery of the diaphragm was found to consist of fairly normal muscular tissue.



1.—Preoperative roentgenogram showing elevation of right diaphragm.

tire diaphragm was incised in the midline, extending from the lateral chest wall
stinum (Fig. 4). This divided the diaphragm into anterior and posterior halves.
thin membrane was then excised, and the two halves of the diaphragm were im-
h interrupted mattress sutures of No. 2-0 black silk. This brought the diaphragm
normal position. The functioning diaphragm now consisted of muscular tissue.
lusion of the operation, rhythmical respiratory contraction of the diaphragm was noted, indicating a functioning phrenic nerve. A small Pezzer catheter was inserted through the seventh interspace for drainage and connected to a water-seal bottle. The chest was closed with two pericostal sutures of No. 2-0 chromic catgut around the eighth and ninth ribs.

The muscles of the chest wall were approximated with continuous No 3 0 chromic catgut, and the skin with continuous No 4 0 silk The baby received 75 c c of blood during surgery and left the operating room in good condition The total operating time was one hour and twenty minutes The infant was placed in an oxygenated crib for twenty four hours The chest drainage catheter was removed in forty eight hours The infant tolerated oral feedings on the second postoperative day The skin sutures were removed on the seventh day A chest roentgenogram taken one week postoperatively shows normal position of the right diaphragm (Fig 2). The patient was discharged from the hospital on May 31, 1953, weighing 9 pounds, 9 ounces (Fig 3)

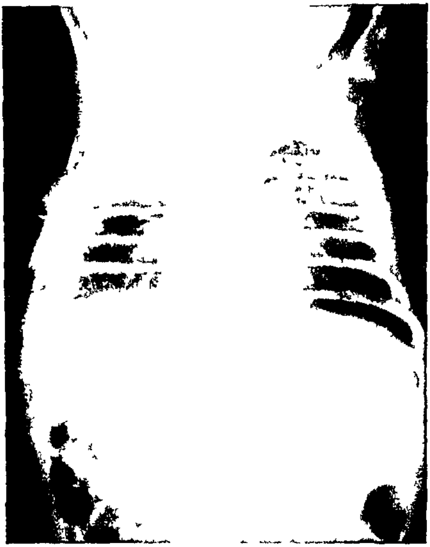


Fig 2—Roentgenogram taken one week postoperatively showing normal position of right diaphragm

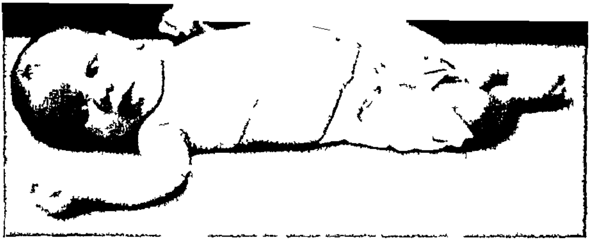


Fig 3—Photograph of infant at the age of six weeks showing operative approach

The infant had complete relief of symptoms following surgery The family moved to Denver, Colorado, a few months after the operation but the pylorus and first part of the duodenum were high and to the right of the midline. The stomach was slow to empty. The remaining laboratory work was within normal limits.

Because of the severity of the symptoms, and failure to gain weight, surgical correction of the diaphragmatic eventration was decided upon.

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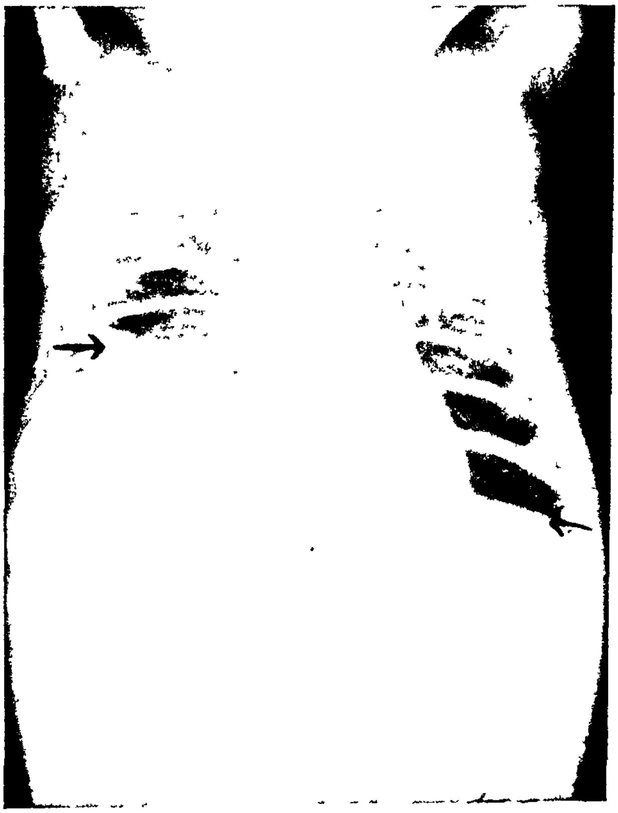

Fig. 1.—Preoperative roentgenogram showing elevation of right diaphragm.

The entire diaphragm was incised in the midline, extending from the lateral chest wall to the mediastinum (Fig. 4). This divided the diaphragm into anterior and posterior halves. The excess thin membrane was then excised, and the two halves of the diaphragm were imbricated with interrupted mattress sutures of No. 2-0 black silk. This brought the diaphragm down to a normal position. The *functioning* diaphragm now consisted of muscular tissue. At the conclusion of the operation, rhythmical respiratory contraction of the diaphragm was noted, indicating a functioning phrenic nerve. A small Pezzer catheter was inserted through the seventh interspace for drainage and connected to a water-seal bottle. The chest was closed with two pericostal sutures of No. 2-0 chromic catgut around the eighth and ninth ribs.

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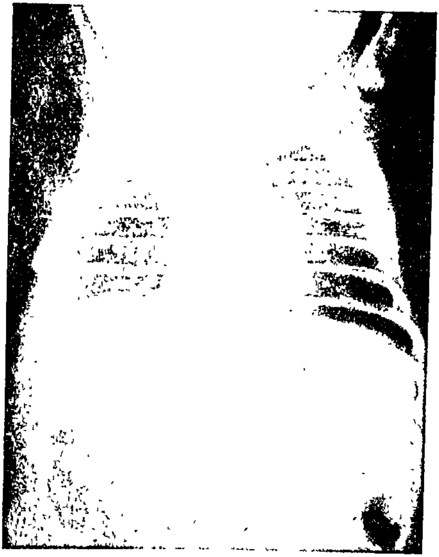

Fig. 2.—Roentgenogram taken one week postoperatively showing normal position of right diaphragm.

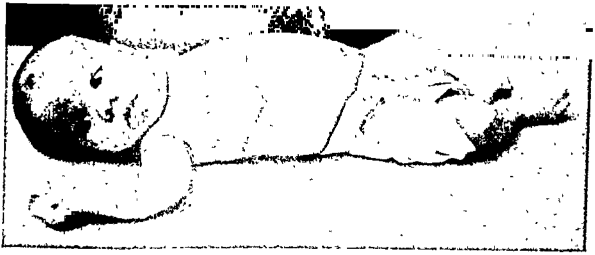

Fig. 3.—Photograph of infant at the age of six weeks showing operative approach.

The infant had complete relief of symptoms following surgery. The family moved to enver, Colorado, a few months after the operation.

Recent communication with the child's mother indicates that he has developed normally and is free from all symptoms. A chest roentgenogram taken at the age of 2 years by the family physician, Dr. M. E. Smernoff of Denver, Colorado, was forwarded to us. This shows maintenance of normal position of the right diaphragm (Fig. 5). The child has had normal physical development during those two years.

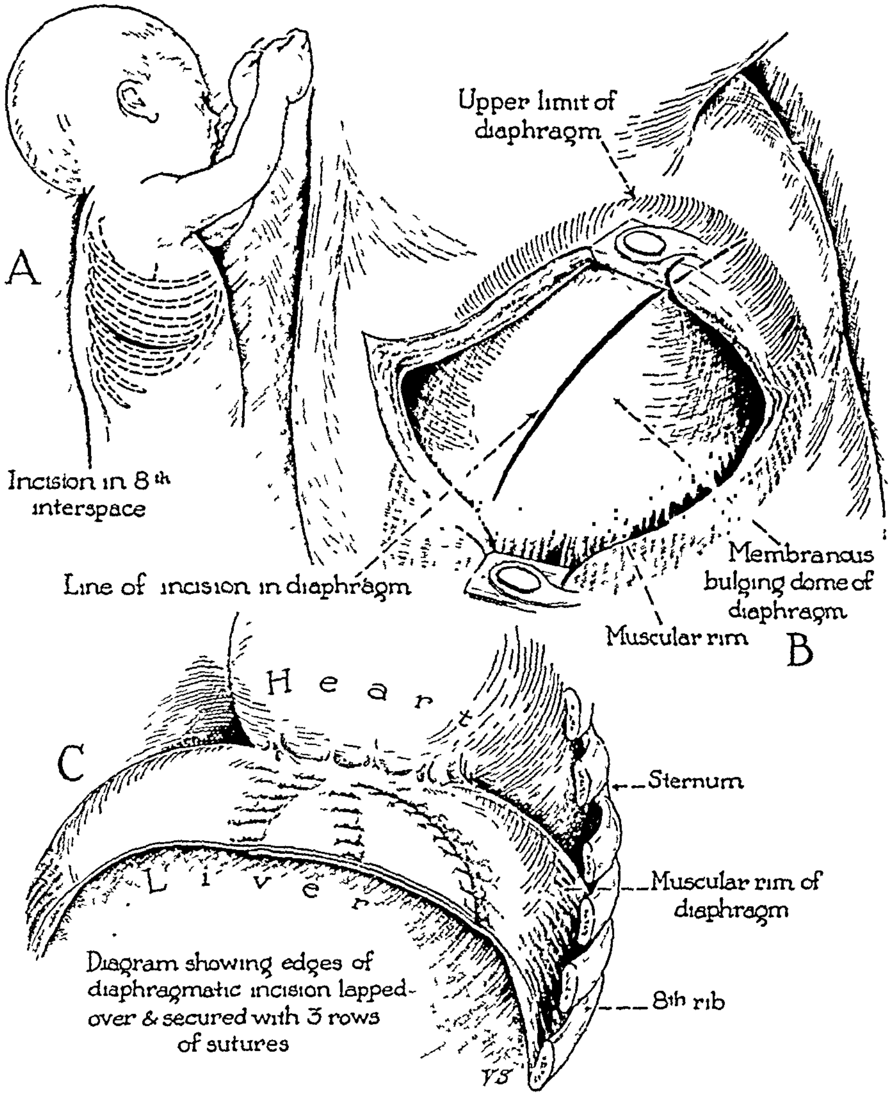
A
Incision in 8th interspace
Upper limit of diaphragm
Line of incision in diaphragm
Membranous bulging dome of diaphragm
Muscular rim
B
Heart
C
Sternum
Muscular rim of diaphragm
Liver
8th rib
Diagram showing edges of diaphragmatic incision lapped-over & secured with 3 rows of sutures
VS

Fig. 1.—Illustration showing technique of surgical repair.

DISCUSSION

The etiology of this defect appears to be a congenital malformation of the diaphragmatic muscle. All of the diaphragm may be involved, but usually it is the central dome.

The intra-abdominal pressure forces the thin membranous diaphragm upward, compressing the overlying lung. There may be a shift of the heart and mediastinum to the opposite side. The above factors may easily produce dyspnea and cyanosis in the young infant.

Eventration of either side may cause angulation of the stomach and pylorus, with partial pyloric obstruction and vomiting. In our case, the pylorus was elevated upward, causing gastric retention and difficulty with feedings.

The diagnosis is usually made by roentgenograms and fluoroscopy. The diaphragm is seen as a thin, curved line, extending high in the pleural cavity. Fluoroscopy reveals slight or no motion on respiration.

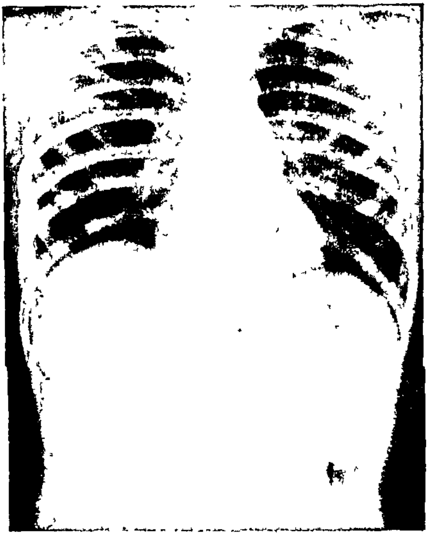

Fig. 5—Roentgenogram taken at the age of two years showing maintenance of position of right diaphragm.

This condition is to be differentiated from diaphragmatic hernia and from phrenic paralysis. If required, barium studies of the gastrointestinal tract should establish the diagnosis of diaphragmatic hernia. It is said that paradoxical motion of the diaphragm can be observed with phrenic paralysis. Quite often there is also brachial plexus paralysis if the trauma is due to childbirth.

Pneumoperitoneum has been used frequently to aid in differential diagnosis in adults; it could also be used in infants if necessary.

Treatment depends on severity of the symptoms. Surgery does not seem necessary for small defects with no symptoms. However, if the eventration is large, with symptoms of dyspnea, cyanosis, cough, and vomiting, immediate surgical repair is indicated. To date, no operative deaths have been reported and the long-term results have been excellent.

Various operative techniques have been described, using both the abdominal and thoracic approach. The usual procedure has been to plicate the thin membranous portion in toward the liver. This portion can then be anchored to the chest wall. Tantalum mesh may be used, as reported by Sanford.[5] In our case, it was decided to excise the membranous portion and to imbricate the two portions of the muscular rim. This procedure gave a strong diaphragmatic repair and did not restrict the motion of the diaphragm.

SUMMARY

1. A case of eventration of the right diaphragm in a six-week-old infant is reported.

2. Successful surgical repair was performed by imbricating the muscular rim of the diaphragm.

3. Symptoms of dyspnea, cyanosis, and cough were completely relieved.

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PREVENTION OF FOAMING IN SUCTION BOTTLES FOLLOWING PULMONARY RESECTION

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THE combination of blood and air bubbling through a water-seal trap after pulmonary resection frequently creates abundant and bothersome frothing. This froth is a nuisance to those engaged in the postoperative care of the patient since bubbles rapidly fill the first drainage bottle and siphon over to the second and third bottles. As a result, the entire suction unit becomes a collection of bloody foam which makes accurate estimation of drainage volume difficult, requires frequent changes of bottles, and is a mechanical hazard to the suction pump.

The addition of a few drops of caprylic alcohol (octyl alcohol, normal-primary) to the first bottle completely prevents any foaming and simplifies this phase of postoperative care.

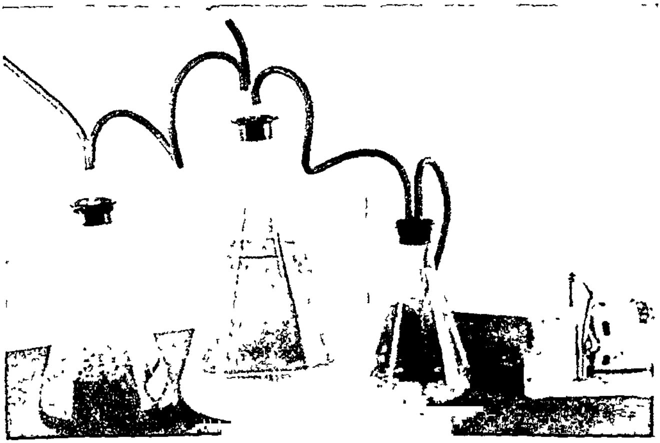

Fig 1—A typical 3-bottle chest suction unit with foaming beginning in the first bottle.

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The opinions or assertions contained herein are those of the authors and are not to be construed as official or reflecting the views of the Navy Department or of the Naval Service at large.

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Caprylic alcohol is a colorless liquid with a pleasant, penetrating, aromatic odor. It is insoluble in water. Its specific gravity is 0.827.[1] Accordingly, the small amount used is highly dispersed, remains on the surface, and will not have access to the submerged portion of the drainage tube communicating with the patient's chest. Hence, any hazard of retrograde aspiration is minimized.

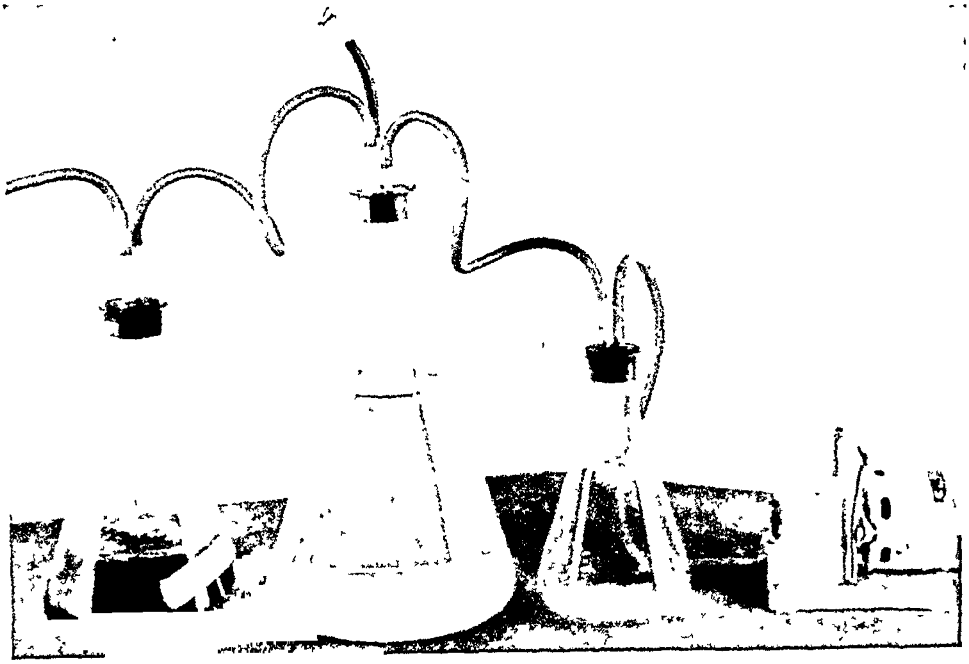

Fig. 2.—Appearance immediately after addition of 0.5 c.c. of caprylic alcohol. All bubbles are immediately destroyed and further foaming is prevented.

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THE REMOVAL OF BENIGN PEDUNCULATED INTRA ATRIAL TUMORS A CLOSED SURGICAL APPROACH

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OUR operative experience with intra atrial tumors at the Bailey Thoracic Clinic *consists of 5 cases In none was the correct diagnosis made pre* operatively In all cases the tumor was encountered during exploration for suspected mitral valve disease, either at our Clinic or at another institution All of our attempts at surgical removal have resulted in mortality except in the last patient operated We were not successful in removing the tumor in its entirety even in the last case However, the operative method used was defeated because of the exceedingly large size of the tumor plus a defect in the instrument used We feel that the tumor in our last case was much larger than will usually be found, if the smaller tumors we had previously encountered can be used as an indication of the average We also feel that the defect in the instrument has now been corrected

Since many patients with suspected mitral valvular disease are now being operated upon at various clinics, more and more surgeons undoubtedly will encounter an occasional intra atrial tumor To date the only report of a successful removal is that of Crafoord,[1] who used hypothermia and an open technique Since it usually is not feasible to induce hypothermia once a closed type of intracardiac operation has been undertaken, it is our hope that *the closed method* we used *in the last patient* will *prove helpful in removing* such tumors

All of the cases of intra atrial tumors that we have encountered occurred within the left atrium, and have been diagnosed as benign myxomas by microscopic examination (Fig 1) In all cases, the pedicle was attached at the rim of the fossa ovalis It was easy to separate the pedicle from its septal attachment by blunt digital dissection These tumors (Fig 2) were soft and friable, and attempts at mechanical removal of them presented the great problem of fragmentation with resulting embolism We, therefore, designed an instrument† which could be inserted into the atrium along the index finger through the purse stringed atrial appendage The end of the instrument contains a band of spring steel to which a meshed nylon bag is attached much like a fish net (Fig *3 A*, *B*, *C*, and *D*) By drawing upon the sliding shaft at the opposite end of the instrument, the diameter of the band's circle is reduced and the mouth of the net becomes puckered so that it can easily be

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†Obtainable from George P Pilling & Son Co Philadelphia P

inserted alongside the operator's intracardiac finger into the atrium (Fig. 4, *A*). After the end of the instrument, plus its attached net, has been passed completely into the atrium, the band is opened to provide a large mouth to the "fish net." The index finger then is used to manipulate the tumor gently into the net (Fig. 4, *B*). After all of the tumor, except its pedicle, has been trapped within the net, the mouth of the net is partially closed so that it is snug around the pedicle. The edge of the band then is pushed against the septum while counter pressure is applied by the finger to the other side of the pedicle (Fig. 4, *C*). Thus, the pedicle is scooped from its attachment to the septal wall and the entire tumor is forced into the net. Then, the mouth of the net is closed tightly. The finger and the instrument are then removed from the heart. As the net is pulled through the atrial appendage (Fig. 4, *D*), the tumor probably will be much larger than the opening in the appendage, but due to its gelatinous consistency it can be easily fragmented, permitting its shape to be altered for easy extraction. Since it is completely enclosed within the net, there is no danger of embolization from such fragmentation.

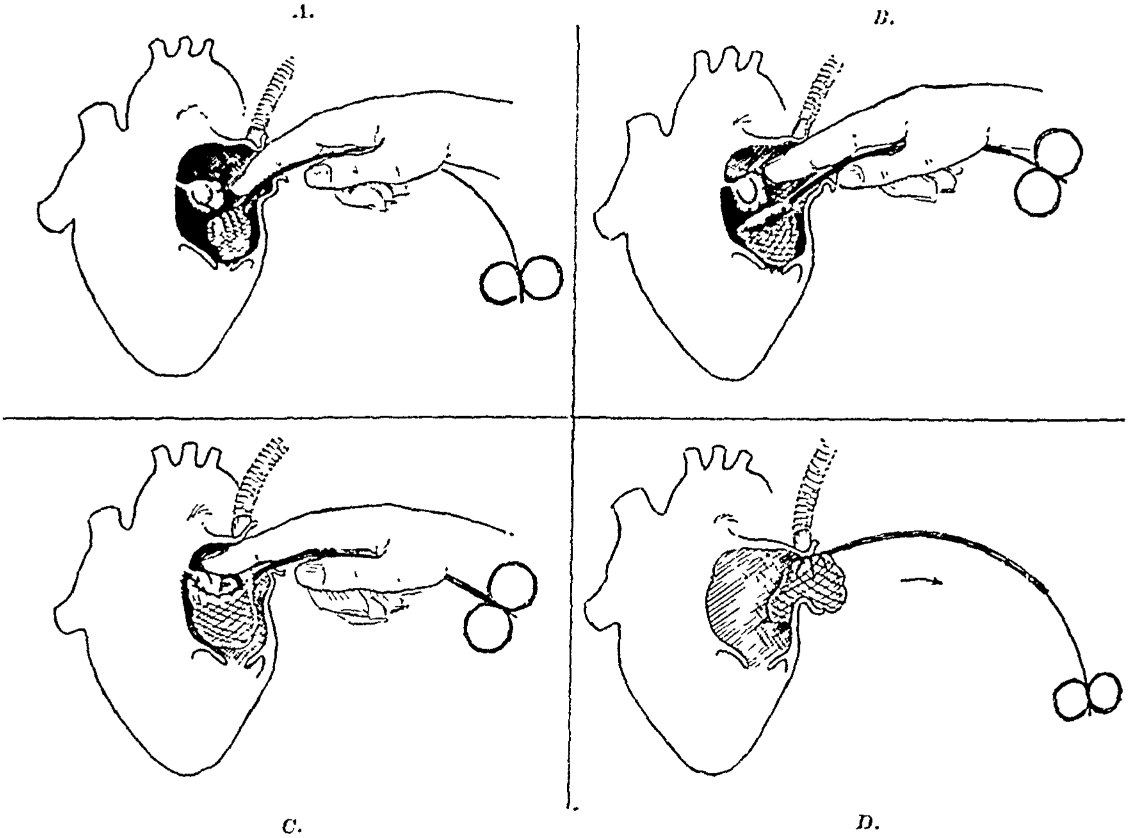

Fig. 4.—*A*. Instrument in place inside atrium with snare closed. *B*, Snare open so that finger can gently manipulate myxoma into "fish net." *C*, Myxoma in "fish net" and pedicle being scooped from attachment at atrial wall. *D*, Myxoma completely enclosed in net and being extracted through opening in atrial appendage.

We utilized the instrument and technique just described on April 5, 1955, at the Hahnemann Hospital, Philadelphia, in a 41-year-old white man (O.S., Hosp. No. 55-03375), who presented symptomatology of increasing exertional dyspnea, nocturnal dyspnea, orthopnea, fatigue, and peripheral edema over a period of twelve years. At the time of surgery, he was no longer able to work. At surgery, the tumor was found to arise from a pedicle attached to the interatrial septum and consisted of multiple lobules which almost completely filled the left atrium. We were able to manipulate a good two thirds of the tumor into the net, but because of its size there was still one lobule, located cephalad and farthest from the mitral valve, which we could not force into the net for fear of fragmentizing it. Thus, we removed that part of the tumor which we could safely net. On extreme tightening of the mouth of the net, the metallic band separated at its welded attachment from the end of the instrument. Therefore, we were not able to use the instrument again to remove the remnant of tumor. Since this unfortunate occurrence, we have redesigned the instrument so that the band is no longer welded to the instrument at one end, but is now a replaceable unit like the wire of a tonsil snare. The weak point of welding is eliminated as a possible breaking point, and even should the band break it can be replaced immediately.

Since we had excised that portion of the tumor lying immediately above the mitral valve, we had hopes that the patient would be improved even by this partial removal. Three months following surgery, the patient had had no recurrence of exertional dyspnea, nocturnal dyspnea, or fatigue. He was able, for the first time in months, to sleep flat in bed, and had returned to work. Since his twelve-year history of increasing symptomatology suggests that the tumor was growing slowly over the same period, we can only hope that he will have several years of freedom from symptoms before requiring further surgery.

SUMMARY

A closed surgical technique for the removal of intra-atrial tumors is presented. Our experience with one operative case in which the major part of the tumor was successfully removed is presented.

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PROCAINE AMIDE IN HYPOTHERMIA

AN EXPERIMENTAL STUDY

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PROCAINE AMIDE has been used clinically in the treatment of auricular and ventricular arrhythmias as, in adequate dosage, it will depress the excitability of both auricular and ventricular muscle.[9] Furthermore, the drug will completely protect the dog heart in situ against electrically induced auricular fibrillation and will also afford a large measure of protection to the ventricles.[8] In certain instances, it has been used in the successful prevention of ventricular fibrillation.[3, 4, 6] The mortality attending the induction of experimental hypothermia has been related to the high incidence of ventricular fibrillation occurring spontaneously during cooling or in the course of circulatory arrest.[1] Although this may be in some degree related to the development of hypercapnia during cooling or to sudden variations in pH levels thereafter,[7] the basic biochemical changes in the myocardial cell are not yet known. Careful electrocardiographic studies in the hypothermic dog indicate that the onset of ventricular fibrillation is preceded by the development of extrasystolic activity although this phase may be so transient that visual appreciation is difficult or impossible. Although unlikely that procaine amide used in such a situation would have any beneficial effect, its trial, both in the prevention and treatment of ventricular fibrillation in the hypothermic dog, seemed warranted.

PRESENT INVESTIGATION

Twenty-five dogs of an average weight of 14 kg. were used in this study, 10 as controls and 15 with procaine amide administered. Under Pentothal sodium* anesthesia, which was also used to control shivering, hypothermia was induced by surface cooling in an ice bath at 4° C. The rectal temperature range obtained was 25 to 30° C. Controlled respiration at a ventilation rate of 24 per minute was maintained by a mechanical respirator using oxygen at 500 ml. per minute in a closed circuit. A soda lime CO_2 absorber was included in the circuit during cooling. Four of the dogs were cooled without caval occlusion. Twenty-one animals underwent a right thoracotomy and caval occlusion with circulatory arrest for periods of 2 to 12 minutes. A right auriculotomy was routinely performed during caval occlusion. Rewarming was carried out to 96° C. by immersion in a water bath at 45° C.

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*The Pentothal sodium used in this study was supplied by Abbott Laboratories, North Chicago, Ill.

Procaine amide was administered by continuous intravenous infusion in concentration of 0.2 Gm. per cent, each dog receiving a minimum of 0.8 Gm. before the commencement of occlusion. In 5 dogs in the treatment of ventricular fibrillation, the drug was given locally into the ventricle or into the arch of the aorta which was clamped above the site of injection, allowing the drug to be perfused through the coronary system by cardiac massage.

RESULTS

Incidence of Ventricular Fibrillation.—No procaine amide. Of the 10 dogs cooled without procaine amide administration, ventricular fibrillation occurred in 4; 1 before caval occlusion, and 3 in the course of caval occlusion with auriculotomy or on release of the caval clamps (Table I). The 6 remaining dogs had caval occlusion periods of from 7 to 10 minutes with normal cardiac function restored. The arterial CO_2 levels of the 9 dogs without fibrillation before circulatory arrest showed considerable variation, as did those of the dog in which fibrillation occurred (Fig. 1).

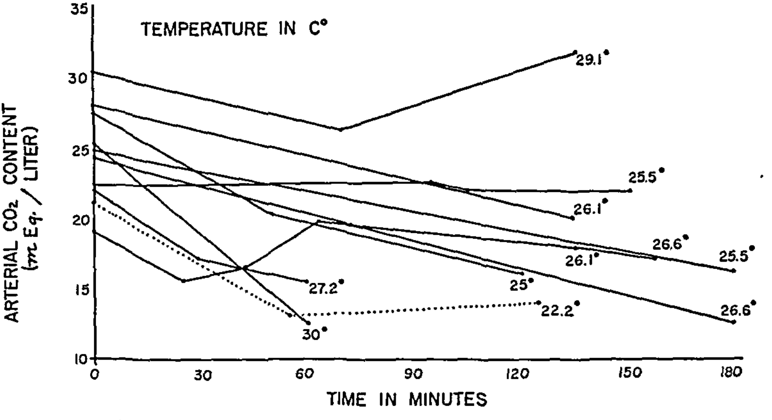

Fig. 1.—Control series of 10 dogs showing variations in arterial CO_2 levels during cooling. No procaine amide was administered. The figures represent lowest temperature reached. All points are before caval occlusion. The dotted-line curve represents the dog in which fibrillation did occur.

TABLE I. INCIDENCE OF VENTRICULAR FIBRILLATION

No procaine amide.—		
Number of dogs	10	
Ventricular fibrillation	4	
a. before caval occlusion		1
b. during caval occlusion		1
c. after caval occlusion		2
With procaine amide.—		
Number of dogs	15	
Ventricular fibrillation	4	
a. before caval occlusion		1
b. during caval occlusion		0
c. after caval occlusion		[illegible]
Ventricular bradycardia and arrest		

Incidence of Ventricular Fibrillation.—With procaine amide. Of 15 dogs with procaine amide administration during cooling, ventricular fibrillation occurred in 1 before caval occlusion. Of the first 6 dogs with caval occlusion and auriculotomy, ventricular fibrillation occurred in 3 on release of the caval clamps, cardiac arrest in 1 after a 12-minute period of caval occlusion, and irreversible bradycardia (8 per min.) in 1 after 9 minutes of occlusion.

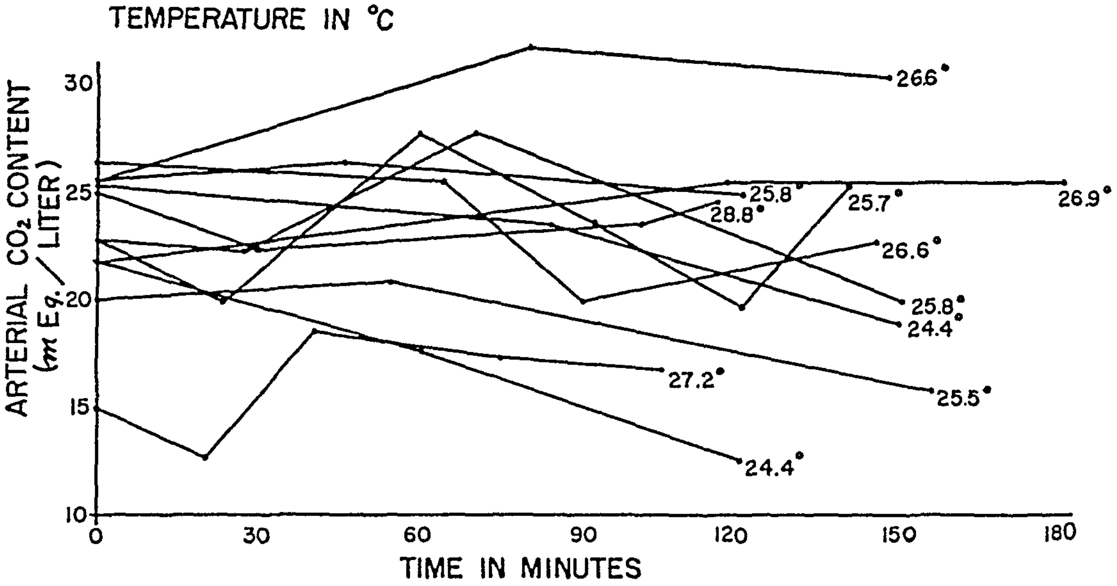

Fig. 2.—Variations in arterial CO_2 levels during cooling with no ventricular fibrillation. Procaine amide was administered.

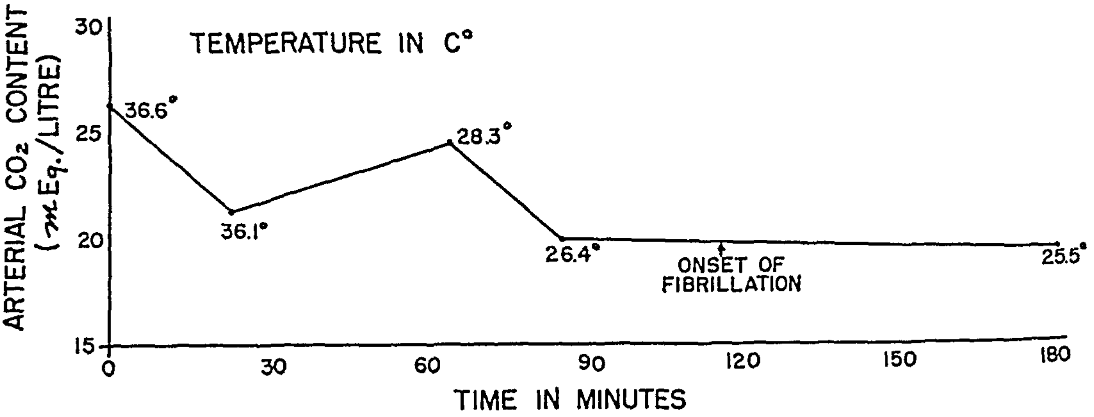

Fig. 3.—Variations in arterial CO_2 levels during cooling with fibrillation occurring despite procaine amide administration. The onset of fibrillation which occurred before occlusion is indicated by the arrow.

Because of these results, the remaining 9 dogs in this group had short circulatory arrest periods of 2 minutes, before resuscitation was undertaken by the use of potassium (Table I). Of the 11 dogs without fibrillation, 5 cases showed wide variations in CO_2 and pH levels at different periods of cooling, while 6 had adequately controlled CO_2 and pH values (Fig. 2). Of the 3 dogs with fibrillation at the release of caval clamps, 1 showed a uniform CO_2 level during cooling, while 2 showed a fall in CO_2 levels (Fig. 3).

Treatment of Ventricular Fibrillation—In the treatment of ventricular fibrillation, procaine amide was injected into the left ventricle and into the aorta to perfuse the coronary system The doses administered were 50, 100, and 150 mg In 5 animals, the drug given in these amounts had no beneficial effect on the existing fibrillation, either clinically or electrocardiographically After 50 mg and 100 mg doses, no change was noted on the electrocardiogram, while in 2 cases, a 150 mg dose slowed the ventricular fibrillation wave, producing a clinical slow "wave" fibrillation (Fig 4) This ventricular activity could not be altered by prolonged massage or by the empirical use of epinephrine or calcium

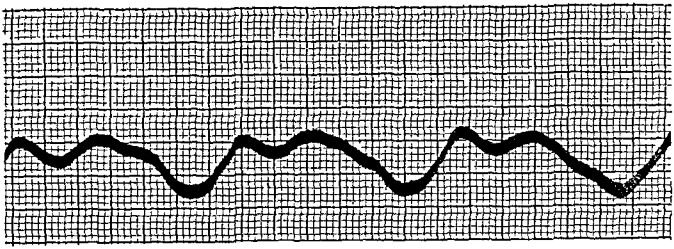

Fig 4—Electrocardiogram (Lead 2) of clinical slow wave fibrillation (25 mm per sec and 1 millivolt per cm)

Cerebral Anoxia—From our experience at the temperature range of 25 to 30° C, 20 minutes has been regarded as the critical limit for circulatory arrest With periods over this time, sufficient cerebral damage apparently occurs to prevent the animal regaining consciousness although reflex and medullary activity returns This period of cerebral anoxia is a summation of the time of caval occlusion and the period from the end of occlusion until the production and maintenance of an arterial pressure of 40 mm Hg or more by cardiac massage

In 6 dogs receiving procaine amide, the period of calculated cerebral anoxia was 5, 8, 12, 15 17, and 19 minutes, respectively These 6 animals did not regain consciousness, living in an unconsciousness state for varying periods up to 48 hours Post mortem examination showed bronchopneumonia in 4 dogs, and no helpful findings in the others The brain was not examined

DISCUSSION

It has become evident from our experience that spontaneous ventricular *fibrillation may occur during cooling in the presence of adequately controlled* CO and, thus, pH levels (Fig 5) Furthermore, in this study, in both control dogs and in dogs receiving procaine amide in which fibrillation did not occur prior to caval occlusion, the CO levels in the majority showed considerable *variation at different periods of the experiment (Figs 2 and 3)* It seems a justifiable conclusion that changes in CO are not the fundamental cause of ventricular fibrillation, although sudden variations may accentuate some causal biochemical shift in regard to the myocardial cell However, procaine amide has not been successful in preventing fibrillation in the presence of changes in CO_2 levels (Fig. 3), and this seems adequate evidence against any assumption that the low incidence of fibrillation during cooling in this study was due to the administration of the drug.

In dogs at normal body temperature, procaine amide in large doses has produced similar effects on the heart to those noted here.[9] In the hypothermic dog, particularly in the presence of circulatory arrest, the effects of the drug are likely to be more marked and more prolonged. Thus, it is of importance that in the treatment of ventricular fibrillation, no slowing of fibrillation has been noted as a prelude to impending slow "wave" fibrillation. In other words, there has been no visual or electrocardiographic evidence of the approaching toxic effect of the drug.

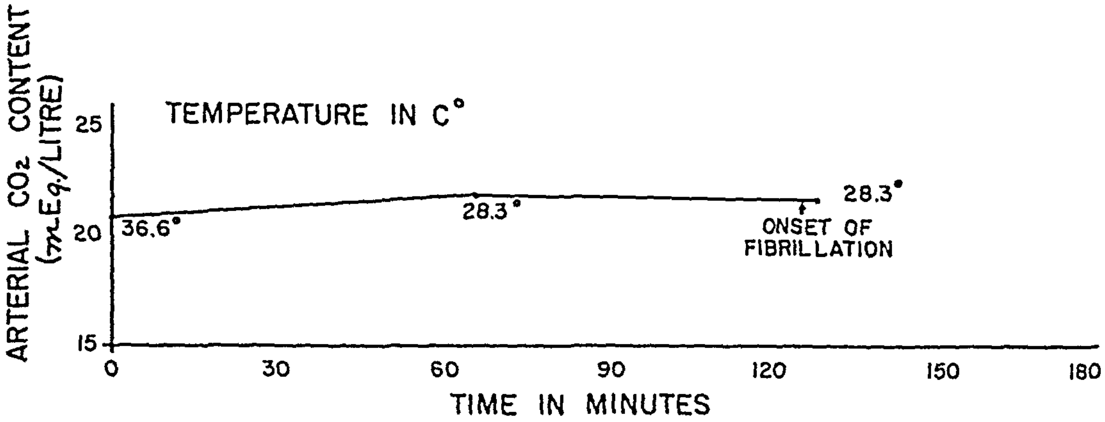

Fig. 5.—Stable arterial CO_2 levels during cooling with fibrillation occurring. No procaine amide was administered. The onset of fibrillation before caval occlusion is indicated by the arrow.

The main effect of procaine amide is a direct one on the auricle. In the denervated heart it depresses sinus action and may eventually produce sinus arrest with nodal rhythm.[9] Although it may also depress the excitability of both auricle and ventricle, its main action in the case of the latter appears to be on diminishing the speed of conduction.[8, 9]

The damage to intraventricular conduction which procaine amide causes may facilitate the re-entry of impulses and the onset of ectopic activity.[9] The circumstance thus arises that procaine amide, which may suppress ventricular automatism when it is established, may lead in the increasing dose to abnormal ventricular activity and fibrillation. With these considerations in mind, it seems unjustifiable to use the drug when circulatory arrest is contemplated, as at this time myocardial ischemia and coronary hypercapnia may already be slowing conduction, and procaine amide may become an additional factor in causing further damage. The position appears to be that as regard to the hypothermic heart, where circulatory arrest is not contemplated, procaine amide will do neither good nor harm, but where occlusion of the inflow tracts is carried out, the use of the drug has serious drawbacks without having, at least experimentally, any obvious advantages.

Cerebral Effects of Procaine Amide.—Although no direct reference could be found in the literature to the effects of hypoxia or hypothermia on the use of procaine amide, there would seem little doubt that both these factors will potentiate the action of the drug. Though less pronounced than those of procaine, the effects of procaine amide on the central nervous system show the same pattern of initial excitation followed by a more prolonged depressant action. In addition to these central effects, depression of systemic blood pressure levels may result from large rapid intravenous doses. Death is usually caused by respiratory arrest.[5, 6] The drug is slowly hydrolyzed in the plasma with 60 per cent excreted unchanged by the kidneys except in the presence of renal impairment or congestive heart failure.[2]

Although in this study procaine amide was infused at a slow rate (between 5 to 7 mg. per minute), the presence of generalized hypothermia combined with circulatory arrest might conceivably produce a cumulative toxic effect as both detoxification and excretion of the drug will be impaired. The 6 dogs in this series with periods of cerebral anoxia of less than the critical limit of 20 minutes did not regain consciousness although reflex activity and spontaneous respiration were present on initial rewarming. Thereafter, death occurred with respiratory failure preceding any evidence of cardiovascular collapse.

SUMMARY

1. Procaine amide, administered by continuous intravenous infusion in concentrations of 0.2 Gm. per cent, did not prevent the occurrence of ventricular fibrillation in 4 dogs out of a group of 15 subjected to circulatory arrest under hypothermia.

2. Intracardiac injection and coronary perfusion of 1 per cent procaine amide solution did not arrest fibrillation, but converted the ventricular myocardium into a nonremedial state of slow conduction which was manifest clinically and electrocardiographically.

3. Presumptive evidence is presented that procaine amide has caused toxic effects on the hypothermic brain resulting in failure of the dogs to regain consciousness, despite the fact that in these dogs the period of cerebral hypoxia in the temperature range 25° to 30° C. was within the known critical limit of 20 minutes.

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NEURILEMMOMA OF THE DIAPHRAGM

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A RAPIDLY increasing scope of thoracic surgical procedures has been reflected by the accruing literature concerning the intriguing and challenging primary tumors and cysts of the diaphragm.[1-6, 9, 12, 15] Particular surgical interest in these lesions was stimulated by the report by Gale and Edwards,[7] in 1939, of a successful excision of a malignant tumor of the diaphragm. By 1950, Samson and Childress[11] accumulated 33 well-authenticated cases of primary tumors from the literature and reported 2 cases of neurofibrosarcoma of the diaphragm, in one of which the tumor was successfully resected. The first primary neurogenic tumor of the diaphragm was apparently reported by Klassen, Patton, and Bemen,[10] in 1945, when they described the successful excision of a large neurofibroma of the diaphragm. Since that time, three other neurogenic tumors have been recorded,[11, 14] two of which were surgically removed. We have not found a previous recorded case of diaphragmatic neurilemmoma, and that fact, in addition to the rarity of the lesion and the interesting clinical picture presented by the patient, has occasioned the following case report.

CASE REPORT

W. B., C. H. 218283, a 55-year-old white housewife, was first seen in consultation on March 24, 1954, because of a severe dry cough and pain in the left chest and arm. The patient had been treated for heart disease and angina for the previous five years. Except for gradually increasing exertional dyspnea there were no other pertinent symptoms and the patient denied hemoptysis, expectoration, and wheezing.

A review of the family history was significant in that the patient's father had died of pulmonary tuberculosis at the age of 40 years and her mother had died at 68 years of heart trouble. Her past history revealed that she had had cholecystectomy performed in 1924, two attacks of left-sided pleurisy, and a tonsillectomy in 1926, and vein ligations of the right leg in 1948.

Physical examination showed a rather apprehensive woman in moderate respiratory distress. Her weight was 167 pounds, the blood pressure 130/90, pulse rate 116 and irregular, and respirations 24. There was dullness to percussion over the right lower chest. The thyroid was moderately enlarged and nodular. Both legs showed a grade 2 pitting edema and varicose veins were present over the right leg. No evidence of digit clubbing was present.

Roentgen and fluoroscopic examinations of the chest and abdomen (Fig. 1) showed a marked density in the left lower chest with an elevated left hemidiaphragm which on normal, slow respiration appeared to move slightly but on sniffing showed a moderate paradoxical movement. Various changes of position including lateral decubitus produced no alteration of the roentgen picture, and studies with and without opaque media demonstrated increased distance between the diaphragm and stomach and colon.

Bronchoscopy was performed under local anesthesia on March 27, 1954. The pertinent findings were a mucoid plug in the left lower lobe bronchus, elevation of that bronchus, and moderate narrowing apparently due to extrinsic pressure. Thoracotomy was con-

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sidered advisable at this time for exploration of an intrathoracic or subdiaphragmatic lesion but because of the patient's general condition and the diagnosis of heart disease with failure, operation was held in abeyance pending further evaluation and study.

In November, 1954, the patient was seen in consultation by one of us (D. M. W.) because of marked cardiac irregularity. The chest pains were noted to be of 12 years' duration, were located in the left subclavicular area and radiated to the deltoid area. They were described as "terrible shooting pains that last for days" which kept the patient from doing her work and were partially relieved by rest and aspirin. There was an associated nonproductive cough attributed to a "bad cold" twelve years previously which had been constantly present since that time except for amelioration during the warm summer months. Paroxysms were precipitated by exposure to cold air and by physical exertion.

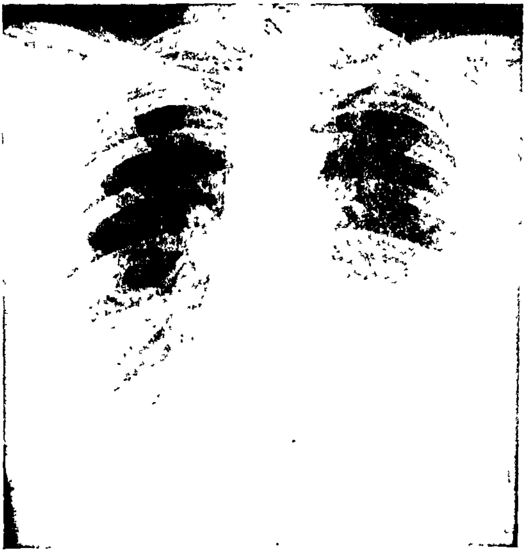

Fig. 1.—Posteroanterior roentgenogram of the chest showing increased density of left lower chest.

Awareness of irregular cardiac action was of four months' duration. It was described as a sensation as though the "heart would jump out of my throat" and was aggravated by activity and dyspnea. Extreme weakness and diminished exercise tolerance were also prominent symptoms.

Functional aspects of her disability included the fact that her mother had "this same kind of a heart cough" for many years before her death. The patient raised six children and two grandchildren and was responsible for the major work on a farm on the outskirts of a small city in which her husband was employed. She acknowledged marked "nervousness," drank a minimum of 12 cups of black coffee daily, and suffered considerably from insomnia.

Examination revealed a depressed, worn-out appearing, stooped, disillusioned woman who appeared older than her stated age. Throughout the interview she sighed a great deal. Her weight was 139½ pounds, the blood pressure 116/72, and pulse was 80 with multiple premature contractions. A moderately enlarged nodular thyroid was present, but no signs of hyperthyroidism could be detected. The heart was normal in size and the heart sounds were normal in character including examination at the base during expiration, and at the apex in the left lateral decubitus after exercise. Physical examination of the lungs revealed dullness and impaired breath sounds at the left base but no evidence of fluid. There was moderate edema limited to the right leg which was the site of extensive varicosities. A 12-lead electrocardiogram revealed multiple premature contractions of ventricular origin, a rate of 58, and no other abnormalities. A teleoroentgenogram revealed no enlargement or displacement of the cardiac silhouette.

Diagnoses were: (1) pulmonary lesion left base of unknown nature; (2) premature contractions of an irritable heart but no objective evidence of organic heart disease; (3) nontoxic nodular goiter; and (4) chronic anxiety tension state aggravated by the consumption of excessive amounts of coffee.

Symptomatic administration of quinidine sulphate and salicylate preparations gave some relief. The patient failed to reduce her intake of coffee and the preponderance of symptoms that appeared to be of functional origin and to be related to a respiratory alkalosis of chronic hyperventilation and to poor posture discouraged exploratory thoracotomy.

She was re-admitted to the hospital on April 1, 1955, because of a marked increase in symptoms of dyspnea, cough, chest pain, palpitations, and swelling of the legs. Physical findings were essentially as described previously except for an increase in the left thoracic dullness and corroborative roentgen findings of an increasing pleural effusion and an acute thrombophlebitis involving the right leg.

Clinical findings of importance were a hemoglobin of 13.5 Gm., the red blood cells numbered 4.54 million, white-blood cells 5,650, and a differential count was within normal limits. The sedimentation rate was 29, the serum bilirubin 0.6 mg. per cent. The total protein was 6.68 Gm. per 100 c.c. of which 3.9 Gm. were albumin and 2.78 Gm. were globulin, or a ratio of 4:3. Examination of 500 c.c. of an amber pleural fluid removed showed a specific gravity of 1.023, a cell count of 10 to 20 red blood cells per high-powered field, 2 to 5 white blood cells of which 11 per cent were polymorphonuclear leukocytes and 89 per cent lymphocytes. No malignant cells were seen in the sediment. Fluoroscopic and roentgen findings were as described previously. Electrocardiographic studies were essentially normal except for premature ventricular beats and the cardiac evaluation at this time was suggestive of no serious organic heart disease. Bronchoscopy was repeated with similar findings to those just described.

A left thoracotomy was performed on April 29, 1955, utilizing a posterolateral incision. The left seventh rib was resected and the pleural space was entered through the periosteal bed. A huge mass was found filling the left lower thorax and compressing and adherent to the lower surface of the left lung and the pericardium. The surface of the mass was covered with large vessels and after careful extensive dissection the tumor was found to arise from a pedicle about 4 inches in diameter in the dome of the left leaf of the diaphragm. The tumor derived its blood supply from the pericardiophrenic and inferior phrenic vessels. The mass was separated from the chest wall, diaphragmatic surface of the lung, and pericardium by sharp and blunt dissection. The central portion of the diaphragm was excised with the tumor and both were then removed. The diaphragmatic defect was repaired by closing the two edges of the defect with interrupted cotton sutures. Hemostasis was secured and the chest wound was closed with intrapleural, subaqueous drainage.

The patient's convalescence was uneventful except for a slow re-expansion of the left lower lobe. She was discharged to her home on May 13, 1955. Follow-up examinations have shown a progressive improvement in the patient's symptoms and general condition. Her heart has been slow and regular without medication and her dyspnea has disappeared. She continues, however, to complain of intermittent left shoulder and chest wall pain but of much less degree than the preoperative pain. Follow-up x-rays have shown a normal postoperative chest (Fig. 2).

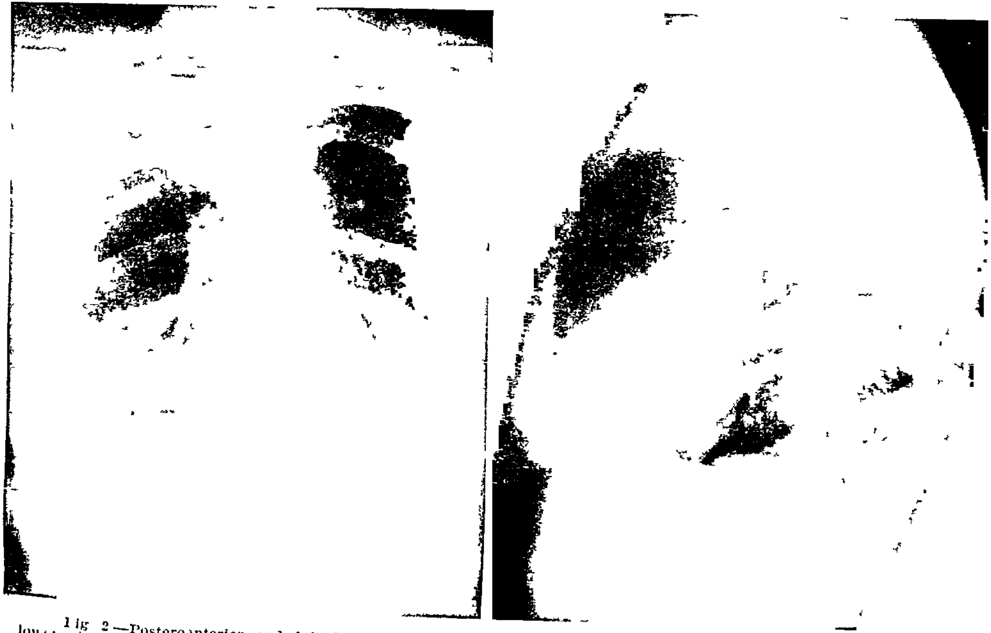

Fig. 2.—Posteroanterior and left lateral roentgenograms of the thorax demonstrating disappearance of left lower chest density, surgical absence of left seventh rib, change in heart silhouette, and postoperative pleural alterations in the left thorax.

PATHOLOGIC DESCRIPTION

Gross Description.—The specimen consisted of a large ovoid tumor which measured 11 by 18 by 23 cm. and weighed 1,390 Gm. (Fig. 3). The external surface presented a variegated appearance. There was apparent encapsulation but many glistening nodules of violaceous, edematous tissue ranging from 0.3 to 5.0 cm. in diameter were scattered over the surface. Small pieces of striated muscle and parts of the central tendon of the diaphragm were on the surface where the pedicle had been dissected free. Areas of the surface between nodules were tan, firm, and quite dense.

The cut surface exposed two dissimilar types of tissue present in approximately equal amounts but irregularly distributed. The solid areas of the specimen were composed of light tan, fibrous, resilient, spheroidal nodules which bulged above a thin membranous pseudocapsule. The resemblance of these nodules, varying from 1.0 cm. to 10.0 cm. in diameter, to uterine leiomyomas was striking. Occasional small yellowish areas were also seen.

The remainder of the cut surface consisted of moist, red-purple, delicately fibrous tissue which contained many small cysts up to 2 cm. in diameter. Direct continuity was seen in many places between the violaceous nodules upon the external surface and similar tissue within the tumor.

Microscopic Examination.—Blocks of tissue were removed from more than thirty-six different areas of the specimen. Paraffin sections were prepared and were stained with hematoxylin and eosin, Gömöri's trichrome stain, Wilder's silver reticulin stain, Verhoeff's elastic stain, van Gieson's connective tissue stain, and with phosphotungstic acid-hematoxylin. Frozen sections were also prepared from representative areas of the fresh specimen and were stained with Terry's stain (polychrome methylene blue), or with Sudan IV for fat.

As noted upon gross examination, microscopy revealed two dominant histologic patterns. Sections from the solid areas were composed of tightly packed, well-differentiated, fusiform or filiform cells arranged in interlacing bundles. Occasionally, the nuclei were palisaded forming ill-defined Verocay bodies. There were no giant cells or mitoses, but scattered foam cells containing sudanophilic vacuoles were noted in some areas. The staining reactions of the tumor cells with the various stains were those of delicate connective tissue (not collagen) rather than smooth muscle (Fig. 4).

The remaining areas of the tumor were very cellular, but the cells were small without defined cytoplasmic borders; the stroma was edematous, almost myxoid, with scattered cysts arising from colliquative necrosis (Fig. 5).

Sections through the pedicle contained tumor, fragments of normal striated muscle, and pieces of the central tendon.

The pathologic diagnosis was benign neurilemmoma arising from the pleural surface of the left leaf of the diaphragm.

DISCUSSION

In his classification of the peripheral nerve tumors, Stout[13] has grouped the neurilemmoma as a benign, neuro-ectodermal neoplasm of supportive tissue type, or a tumor of nerve sheath origin. He lists as synonyms of this tumor the following: neurilemmoma, angioneurofibroma, false neuroma, fibroganglioma, acoustic neuroma, fibromyxoma, glioma, myoschwannoma, neurilemoblastoma, neurinoma, neurofibromyoma, neuroma fibrillare, peripheral glioma, perineural fibroblastoma, perineural glioma, Schwannoma, Schwannoglioma and specific nerve sheath tumor.

It is obvious from this foregoing list of synonyms that lesions of this type have been described by numerous authors and have been found in many locations, including the thorax.[8] Most of the gross descriptions of tumors have

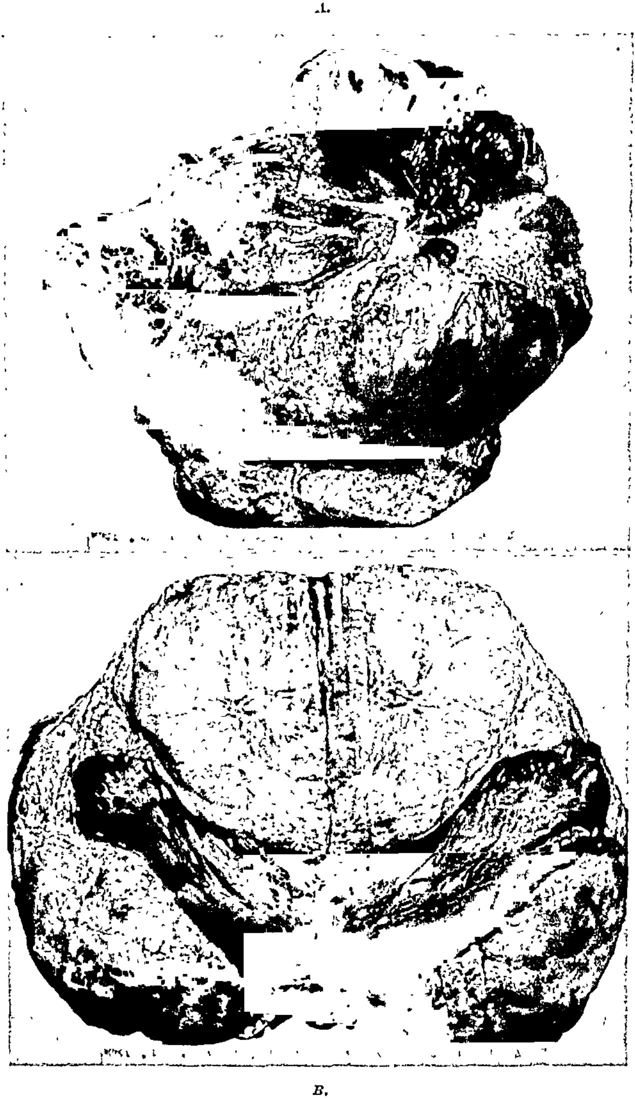

Fig. 3.—*A*, External appearance of tumor showing irregular surface with large and small cysts, nodules, and dilated blood vessels. *B*, Cut surface of tumor demonstrating lobular division of tumor by mucoid septa.

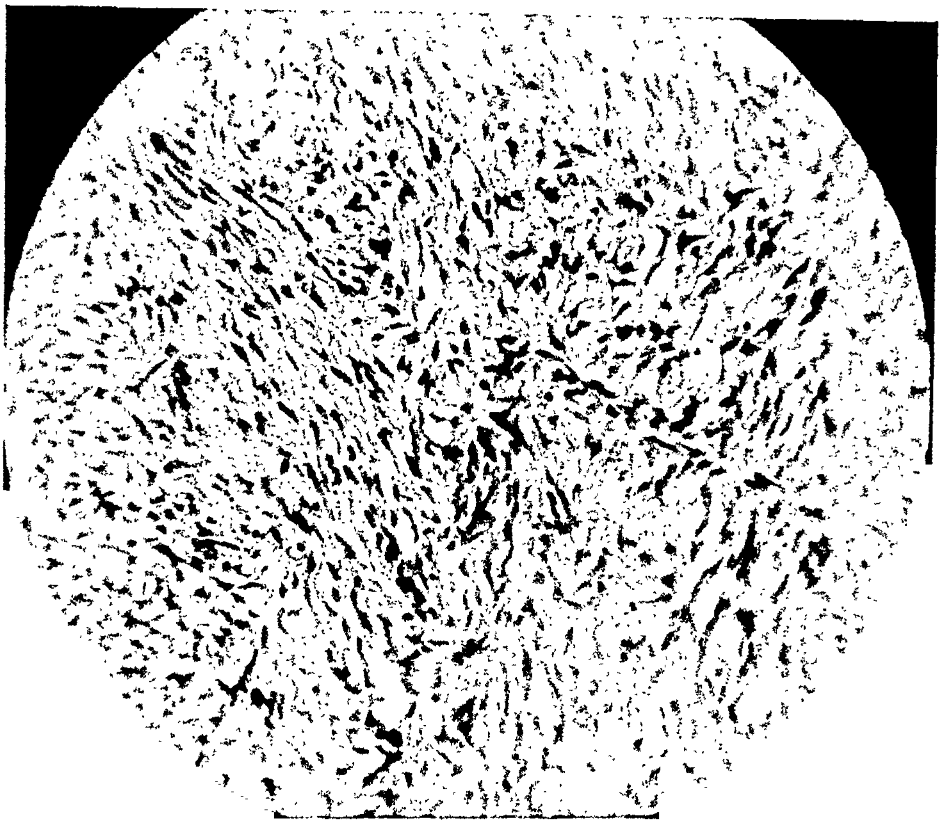

Fig. 4.—Microscopic section demonstrating Antoni type A pattern with well-differentiated filiform and fusiform cells, interlacing bundles, and palisading nuclei (X75).

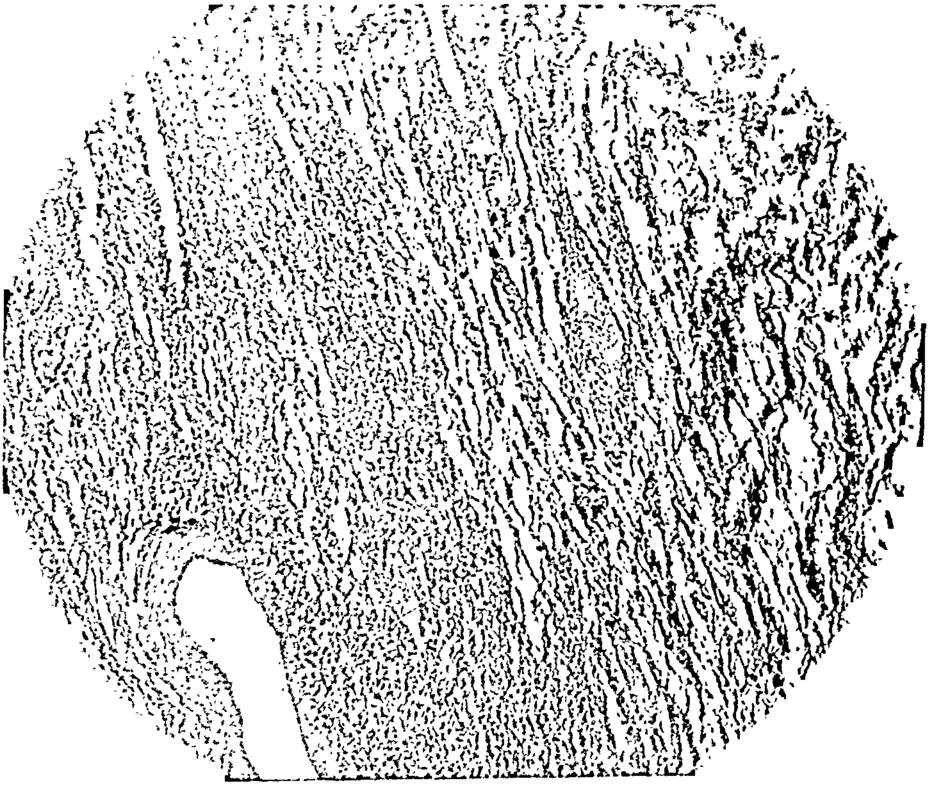

Fig. 5.—Histologic section of Antoni type B area, showing cellularity, myxoid stroma, large nuclei, and haphazard tissue arrangement (X45).

corresponded to that reported herein and the encapsulated variety has been considered most frequently to be benign. The histologic picture has been characteristic and two microscopic patterns consisting of a solid complex, the Antoni type A, and a cystic or loose complex, the Antoni type B, have been the most common.[13]

The symptoms and signs exhibited by this patient have been similar to those previously described.. The shoulder and arm pains were apparently due to diaphragmatic irritation. The cough could have been due to pleural and *bronchial derangement, while the exertional dyspnea could well have been the* result of mechanical interference with respirations.

The diagnosis of this tumor was dependent on the surgical exploration of the thorax, and although pneumothorax and pneumoperitoneum were not performed, it was not felt that they would have added to the roentgen survey described. In retrospective consideration, the patient should have been operated upon sooner and would have been if the nature of her disability had been understood and the functional aspects of her disability had created less of a problem.

The patient's postoperative course has been singularly favorable and, in view of the benign character of the lesion, an excellent prognosis should be her outlook.

SUMMARY

1. A primary neurilemmoma of the left leaf of the diaphragm occurring in a 55-year-old housewife is presented.
2. The lesion was successfully resected with recovery of the patient.

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HYPOTENSION FROM ARFONAD: AN AID IN THE SURGICAL TREATMENT OF PATENT DUCTUS ARTERIOSUS IN OLDER PATIENTS

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A METHOD of producing hypotension from Arfonad makes the surgical treatment of patent ductus arteriosus in aged patients more safe.

Hypotension is easily controlled by a single anesthetist by regulating an intravenous drip of a solution containing Arfonad. Controlled hypotension causes the vessels to become flaccid and more easily and safely manipulated. Hypotension produced in this manner by Arfonad is without apparent serious side effects.

This type of surgery has been more difficult in older patients than in children because of loss of elasticity of large vessels, atheromatous changes, loss of tubular structure of the ductus, and a higher blood pressure. The combination of these conditions make it technically more difficult to divide and suture the patent ductus arteriosus in patients in the third and fourth decades.

Groves and associates have used hypotension produced by the method of Gardner and Hale at the Cleveland Clinic Foundation to aid in the surgical treatment of a large or complicated patent ductus arteriosus.[1] This method of lowering the blood pressure is performed by bleeding the patient until the desired state of hypotension is reached. The blood is taken from a peripheral artery and is replaced in a similar manner as necessary at the termination of the procedure. This method has much to commend it, but is a little too cumbersome to use for hypotension needed for only short periods of time. The procedure also necessitates a trained individual besides the anesthesiologist to aid in regulating the level of hypotension. Equipment is necessary to replace the blood under pressure and care must be exercised to avoid air embolism. Finally, an artery must be utilized for the procedure, which is difficult to cannulate without open exposure of the vessel.

Hypotension produced by drugs obviates these difficulties. Arfonad appears to be an ideal drug for lowering the blood pressure and it has been used in many types of surgery as indicated in a report by Sadove, Wyant, and Gleave.[3] Arfonad is a thiophanium derivative which has the characteristic ganglionic blocking and hypotensive effects of tetraethylammonium bromide.[2, 3]

I have observed a neurosurgical colleague use hypotension produced by Arfonad to facilitate ligation of an aneurysm on a middle cerebral artery. The procedure was striking in that the vessels were seen to pulsate feebly and appeared to have little tension. The ease of handling this type of aneurysm was

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The Arfonad used in most of the cases reported in this series was furnished through the kind cooperation of Dr. Thomas C. Fleming of Hoffmann-La Roche, Inc.

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remarkable in comparison to those treated with normal arterial pressure Of course, the application of this type of hypotension to large vessel surgery was immediately suggested

CASE REPORTS

CASE 1—R W., a white woman, aged 30, complained of shortness of breath, fatigue, and rapid heart action She had had a febrile illness at the age of five or six which lasted for several weeks, and some years later was said to be rheumatic fever In high school, a heart murmur prevented her from entering in competitive sports In 1945, an illness, of which she did not remember the details or symptoms, kept her in bed six weeks Since then she followed a restricted life, but she had had three pregnancies that terminated in normal deliveries without complications For one year, there had been a noticeable increase in shortness of breath with mild exertion

Physical examination showed the pulse to be 90 and blood pressure 120/40 A continuous precordial murmur was present which was loudest in the second left interspace and infraclavicular areas The heart was active and there was a diffuse apical impulse There was some cardiac enlargement with the left border of the heart outside the midclavicular line

A roentgenogram of the chest revealed a prominent pulmonary artery and accentuation of the vascular markings of the lungs The heart was moderately enlarged with a rounded apex

An electrocardiogram revealed some myocardial changes, but nothing of diagnostic significance, there was no axis deviation

Cardiac catheterization revealed a pulmonary artery pressure of 80/40 The right ventricular pressure was 80/0 8 Oxygen saturation studies were consistent with the diagnosis of patent ductus arteriosus

On Feb 16, 1954, division and suture of a ductus, 1 cm in diameter, was performed This was facilitated by lowering the systemic pressure to 80 mm by the use of Arfonad for a period of about fifteen minutes The pressure was allowed to return to normal after the clamps were removed and no bleeding was noted from the suture lines

The postoperative course was uneventful and an examination on March 24, 1954, revealed a 1 5 cm reduction in the transverse diameter of the heart No murmurs were evident

CASE 2—P N, a white woman, aged 38, complained of fatigue and mild exertional dyspnea A heart murmur since birth restricted her activity. During the last few years she had noted increasing fatigue and some shortness of breath while doing her housework.

Physical examination revealed a blood pressure of 135/55 and a regular pulse of 84. A continuous murmur with systolic accentuation was present at the base of the heart with the point of maximum intensity in the second left interspace. The pulmonic second sound was accentuated The remainder of the physical examination was essentially normal

A roentgenogram of the chest showed some left ventricular enlargement and prominence of the pulmonary artery

Cardiac catheterization studies showed a pulmonary artery pressure of 17 8/10 12 Pressures in the right ventricle were 22 24/2 5. Oxygen samples were consistent with the diagnosis of patent ductus arteriosus

On March 4, 1954, division and suture of the patent ductus arteriosus was performed The ductus measured 1 4 cm. in outside diameter and was very short After the ductus had been dissected, Afronad was given and the systemic pressure was lowered from 140 mm to 90 mm. systolic The ductus was clamped, transected, and sutured uneventfully. It was of interest to note that the wall was rather sclerotic and irregularly thickened. The pressure was allowed to return to the normal level after the clamps had been removed (about fifteen minutes total time)

The postoperative course was uneventful and one month later an examination revealed a very satisfactory appearance. The heart size had diminished 1 cm. in transverse diameter as noted on a roentgenogram of the chest.

Case 3.—F. L., a white woman, aged 23, was first seen in St. Anthony's Hospital because of toxemia of pregnancy and spontaneous abortion. At the time of this hospitalization, enlargement of the heart and a heart murmur were noted. In reviewing the cardiorespiratory system, it was found she had noted forceful heart action for many years but had not given this much thought. She was certain it had become gradually worse and on occasions at night this was sufficient to shake the bed. This forceful beating was accentuated by exercise which also caused shortness of breath. No other significant symptoms were elicited.

Her history revealed severe pneumonia in the first year of life with near fatal outcome. Recurrent respiratory infections had occurred every year and frequent bouts of epistaxis had been present from the age of six years.

Physical examination showed a very slender girl, taller than average. The blood pressure was 150/50, pulse 88, temperature 98.6° F. The carotid arteries pulsated vigorously and there was a slight Corrigan pulse present in the extremities. The heart appeared to be moderately enlarged. A loud harsh systolic murmur was present which continued through the second sound and ended in early diastole. This murmur was loudest over the pulmonic area and left sternal border. P_2 and M_1 were accentuated.

Fluoroscopy of the chest revealed a prominent and somewhat dilated pulmonary artery with rather marked pulsation of the pulmonary artery and hilar vessels. The left ventricle was markedly enlarged.

Electrocardiograms showed hypertrophy of the left ventricle. Cardiac catheterization studies were consistent with a patent ductus arteriosus. The pulmonary artery pressure was 64/40 mm. Hg and a right ventricular pressure was 70/5 mm. Hg.

She was admitted to St. Anthony's Hospital Feb. 12, 1954, and division and suture of the patent ductus was carried out. During clamping, cutting, and suturing of the ductus arterious, Arfonad was administered to lower the systemic blood pressure to 80 mm. systolic. The ductus was conical in shape, measured 1.5 cm. in diameter on the pulmonary artery aspect, and was much wider on the aortic side.

Convalescence was uneventful and a follow-up examination in four months revealed a blood pressure of 118/74. No murmur was evident and the heart was quiet compared to the preoperative examination. The pulse was no longer of a Corrigan type and the carotid arteries did not pulsate markedly.

Case 4.—J. B., a white woman, aged 38, had known patent ductus arteriosus for at least ten years, but with few symptoms. Six months prior to the referral by her physician, a severe respiratory infection occurred which responded to antibiotics. A cough persisted and was productive of mucoid sputum. The cough was paroxysmal and occasionally the sputum was streaked with blood. About ten days prior to hospital entry, a second respiratory infection occurred with aggravation of the cough. She smoked a package of cigarettes a day which seemed to aggravate the cough.

Physical examination revealed a pulse of 80, blood pressure 130/60. A continuous murmur typical of patent ductus arteriosus was present over the second left interspace and infraclavicular area. An occasional sibilant râle was present.

Roentgenograms of the chest revealed marked prominence of the pulmonary vessels. The transverse diameter of the heart measured 15.5 cm. as compared to a 28 cm. transverse diameter of the thorax.

On May 3, 1955, division and suture of the patent ductus arteriosus was performed with the aid of hypotension induced by Arfonad. The ductus arteriosus measured 1 cm. in diameter on the pulmonary aspect. The intima presented partially calcified plaques.

She was discharged on the tenth postoperative day after an uneventful postoperative course. A follow-up roentgenogram in one month showed some reduction in the size of the heart.

Case 5.—L. D., a white woman, aged 38, was known to have a heart murmur since an early age. Whooping cough had occurred at the age of six weeks during which there was some cyanosis. After that, she had no further episodes of cyanosis. An occasional irregularity of the heart had been noted. Coughing began about eight weeks before admission to St. Anthony Hospital on Jan. 15, 1955. She denied restriction of activity at any time.

Physical examination revealed a pulse of 84, blood pressure 144/70. The heart examination showed a continuous murmur over the second left interspace and infraclavicular areas. Moist and dry rales were audible over both bases.

Arrangements were made for hospital admission, but acute respiratory distress occurred necessitating an emergency admission. There were coarse sibilant rales throughout the lung fields and prolongation of both phases of respiration. It was thought these symptoms were due to an acute obstructive bronchitis superimposed on pulmonary hypertension.

Medical therapy was effective in clearing the respiratory infection and on Jan. 26, 1955, division and suture of the patent ductus arteriosus was performed. The vessel measured 1 cm. in diameter on the pulmonary aspect. Arfonad was used to cause hypotension during the clamping, cutting, and suturing of the ductus. The pressures were brought back to normal during the suturing of the pulmonary side. When the Gelfoam pledget was removed from the aortic suture line, bleeding occurred which necessitated two interrupted sutures for control.

The patient was discharged from the hospital on the eleventh postoperative day. Examination in three weeks revealed a pulsus alternans which disappeared with digitalis administrations. The heart had diminished in size slightly. The subsequent course was uneventful.

DISCUSSION

The changes in the character of the vessels with a drop of systolic blood pressure of 30 to 40 mm. Hg is remarkable. The aorta, which is tense and often bulbous at the site of ductus arteriosus, becomes pliable and almost flaccid and has a feeling of elasticity that is not present prior to the production of hypotension. Clamping, cutting, and suturing of the ductus is done with about the same ease as in a child. Bleeding is minimal from the suture line at the time the clamps are released and as the pressure is brought back to normal levels, no further bleeding occurs.

The pressure is allowed to remain down only during the time of actual clamping, transection, and suturing of the ductus, a matter of some fifteen to twenty minutes, at the most. This time, with the systolic pressure of around 80 mm., is not sufficient to be injurious to the vital organs, a matter for consideration in prolonged procedures. Clinical shock is not evident as the pulse rate remains at the prehypotensive rate.

Arfonad is usually administered intravenously in a 0.1 per cent concentration. The blood pressure drops slowly, at times taking as long as five minutes to reach the desired state of hypotension. The pressure rises slowly after the drug is discontinued. Neosynephrine or a similar vasopressor drug will cause an immediate rise in pressure should the need occur. However, after the administration of a vasopressor agent, it is almost impossible to lower the pressure again by further administration of Arfonad. This point is of practical consideration for vasopressor agents should not be given anytime prior to the use of Arfonad, if this type of hypotension is contemplated.

An outstanding feature is the facility with which the pressure can be controlled by an unaided anesthesiologist. Control is possible by regulating the rate of intravenous flow of fluid containing the drug. As the need for hypotension is very short, this method is particularly applicable to the division and suture of a patent ductus arteriosus. Complicated problems such as an aneursym of the pulmonary artery, in cases of patent ductus arteriosus, might also be more easily treated with hypotension produced by Arfonad.

SUMMARY

1. More technical difficulties occur in the surgery of patent ductus arteriosus in older patients than in children, and the risk is thereby increased.
2. These technical difficulties are directly related to : (a) loss of elasticity of the large vessels and to atheromatous changes; (b) loss of the tubular structure of the ductus; and (c) a relatively higher pulmonary artery pressure.
3. Controlled hypotension causes the vessels to become flaccid and more easily and safely manipulated.
4. Hypotension is easily controlled by a single anesthetist by the regulation of an intravenous drip of a solution containing Arfonad.
5. Hypotension produced by Arfonad is without apparent serious side effects. Hypotension of 80 mm. Hg systolic for 15 to 20 minutes is sufficiently long for division and suture of the patent ductus arteriosus and of not sufficient duration to be injurious to vital organs.
6. A slow reversal of hypotension is obtained by stopping the administration of the drug and a more rapid reversal is caused by the administration of a vasopressor agent.
7. Five case reports of patent ductus arteriosus so treated are presented.

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PARTIAL RESECTION OF THE CLAVICLE IN THE SURGERY OF PULMONARY TUBERCULOSIS

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VARIOUS methods of dealing with large chronic tuberculous bronchopleural fistulas, empyemas, and subscapular space infections have gained acceptance among chest surgeons over the past few decades. These include extrapleural and Schede thoracoplasties, muscle transplantation, partial scapulectomy, and skin grafting. Occasionally a direct resuture of the open bronchus is said to be successful, and sometimes re-resection has to be done. The problem of obliterating chronically infected tuberculous spaces, either intra- or extrapleural, is complicated by the rigidity of the bony structures of the chest wall and the shoulder girdle. Usually, when a sufficient number of ribs has been removed, overlying the infected space, or a sufficiently large portion of the entire thickness of the chest wall, including ribs, intercostal muscles, and parietal pleura, has been resected, closure of an empyema cavity can be achieved. Similarly, excision of infected material, partial scapulectomy, and muscle transplantation are usually effective in obliterating a subscapular space infection. Occasionally, however, the infected space persists, especially in its uppermost extremity, in spite of all efforts to obliterate it. In two such instances it occurred to us that the muscles of the shoulder girdle could not fall in against the chest wall, in the case of a subscapular infection following thoracoplasty, or against the visceral pleura and mediastinum, in the case of an apical empyema with large bronchopleural fistulas, because the shoulder girdle was held out from the chest wall by the rigid clavicle. This seemed to prevent obliteration of the infected spaces in each instance. It was decided that resection of a portion of the clavicle, in combination with other procedures, might result in sufficient medial displacement of the entire shoulder girdle to allow healing to take place. Since, to our surprise, both cases were successful, we are reporting them in some detail.

CASE REPORTS

CASE 1.—G. S. K., a 22 year old Indian boy, was first admitted to the Wanless Sanatorium in 1950 with far advanced chronic pulmonary tuberculosis of three years duration. He had a large cavity in the left upper lobe, which left artificial pneumothorax had failed to control in spite of a partial internal pneumonolysis. Consequently, a two stage left posterolateral

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From the Wanless Tuberculosis Sanatorium, Wanlesswadi, S. Satara, India.

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thoracoplasty was done, and completed in October, 1951. The wound became infected, and a chronically draining sinus from a subscapular space infection persisted for three years. This sinus, which opened in the lower third of the old posterolateral skin incision, was partially excised or curetted on four separate occasions over the ensuing years, but after remaining closed for a few weeks, it reopened each time. He was readmitted for the third time in March, 1954, and a Lipiodol study of the space showed that it extended well up behind the left clavicle (Fig. 1). The sputum smears for acid-fast bacilli had been negative for years. Culture of secretions from the sinus was not available.

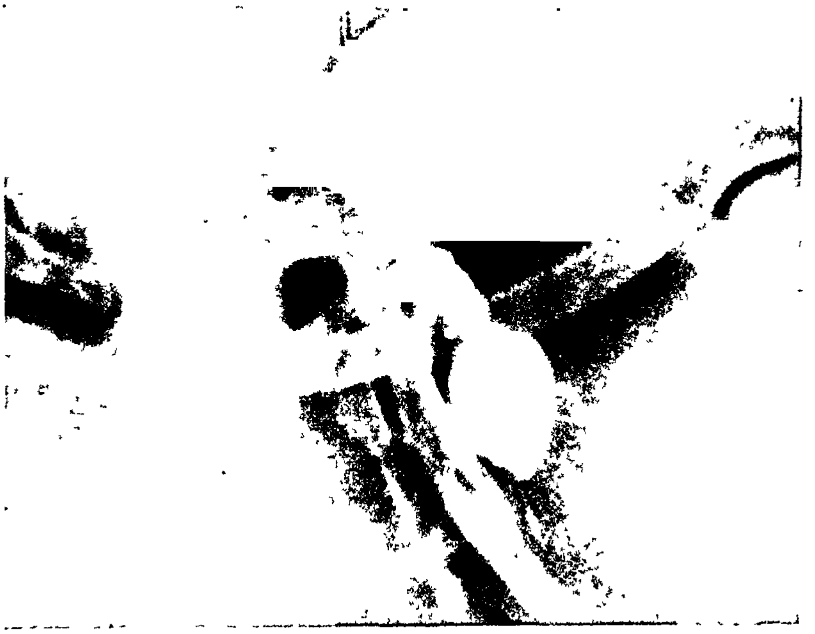


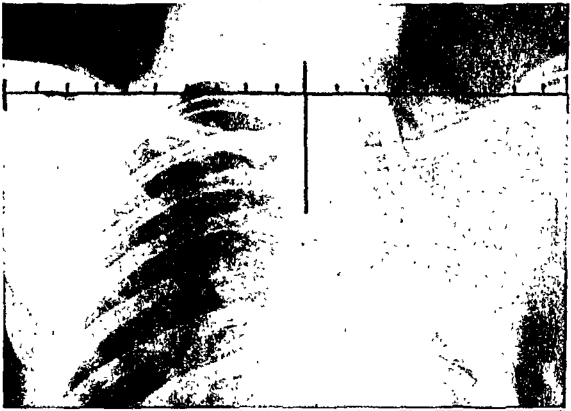
Fig. 1.—Case 1. The chronically infected subscapular space, outlined by Lipiodol, extends well up behind the clavicle.

On March 5, 1954, under Xylocaine epidural block, with the patient lying on the right side, the old scar, sinus tract, and infected space were widely excised. The regenerated bone which formed a wall of the space was quite healthy looking, and no foreign body was found. One quickly got the impression that the large remaining dead space was being held open by the strutlike action of the left clavicle, and that a portion of this would need to be resected. Accordingly, the wound posteriorly was closed around a large rubber tube drain, and the patient was turned over on his back. Using fresh gowns, gloves, and instruments, a 6 cm. portion of the middle of the left clavicle was subperiosteally excised, and the wound closed without drainage. Pathologic examination of the excised posterior sinus tract showed chronic granulation tissue consistent with tuberculosis. Culture was not made.

For two weeks following surgery the patient was given penicillin and streptomycin. The drain was removed on the third postoperative day. The wounds healed completely, and have remained healed for the twenty months since operation that he has been followed. The degree of medial displacement of the left shoulder girdle as a result of the operation, is illustrated by Figs. 2 and 3.

Case 2.—Y. B. I., a 33-year-old male Arabian patient, was admitted to the Wanless Sanatorium in June, 1951, with far advanced pulmonary tuberculosis, bilateral, of about six months' duration. He had had a 5-rib two-stage right posterolateral thoracoplasty performed in September, 1952, but the sputum remained positive for acid-fast bacilli, and a residual cavity was apparent under the thoracoplasty on radiograms. On March 12, 1954, therefore, right upper and middle lobectomy, and resection of the superior segment of the right lower lobe were carried out. The postoperative course was stormy, with the development of recurring atelectasis of the basal segments of the right lower lobe. Eventually the patient refused further bronchoscopies and intratracheal aspirations, and a tracheostomy had to be done to aspirate the right lower lobe. Nevertheless, an empyema and multiple bronchopleural fistulas developed, and open drainage was established three weeks after the resection. In May, 1954, the first of two stages of Schede thoracoplasty was carried out, with resection of the regenerated bony plate from the old thoracoplasty, including the

Fig. 2.



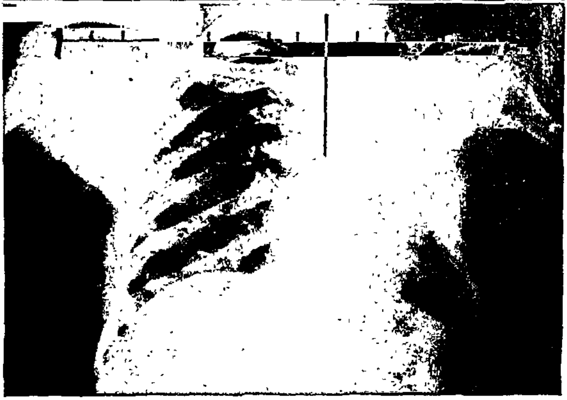


Fig. 3.

Fig. 2.—Case 1. Chest x-ray before claviculectomy. Each division equals 1.5 cm. The air-filled subscapular space is clearly seen on the left side.

Fig. 3.—Case 1. Chest x-ray following resection of 6 cm. of the middle portion of the left clavicle, showing medial displacement of entire left shoulder girdle, and obliteration of the infected subscapular space.

first five ribs, intercostal muscles, and infected parietal pleura. Two months later the second stage was done, including the seventh through the tenth ribs and underlying structures. A wide open wound remained, with infected costal cartilages in its anterior depths, a collapsed lower lobe inferiorly, and multiple bronchopleural fistulas superiorly. The scapular musculature could not come medially against the mediastinal pleura because it seemed to be held out by the strutlike right clavicular bone. Accordingly, partial claviculectomy was decided upon and carried out in August, 1954, five months after the original resection. At the same time, after carefully draping away the clavicular wound, multiple infected costal cartilages were resected through a separate parasternally placed

Fig. 4.

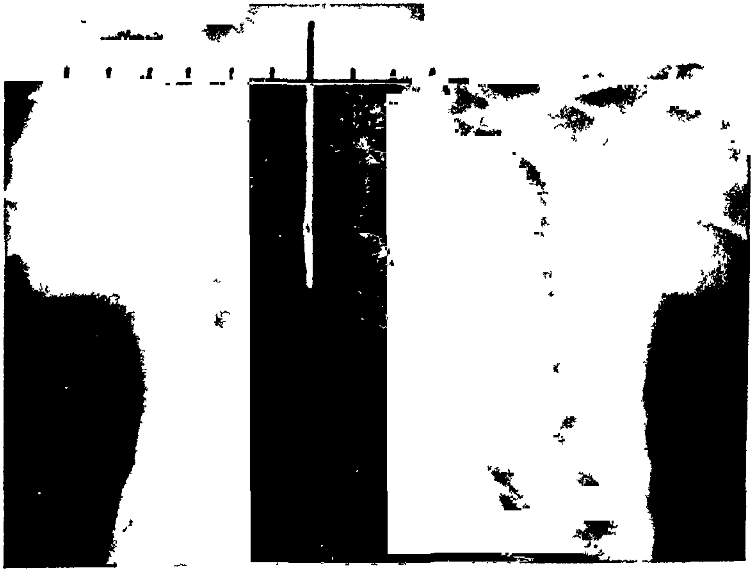


Fig. 5.

Fig. 4.—Case 2. Chest x-ray prior to partial resection of the middle portion of the right clavicle. A tracheostomy tube is in place. (See text.)

Fig. 5.—Case 2. Chest x-ray following partial resection of the right clavicle, showing medial displacement of the entire right shoulder girdle and obliteration of empyema space.

incision A second removal of parasternal infected costal cartilages was done two months later but, by this time, it was apparent that the shoulder girdle was well up against the mediastinal pleura, and that closure could be accomplished of the long narrow but deep posterolateral wound, in the bottom of which three bronchopleural fistulas still were present, though considerably smaller In January, 1955, ten months after the resection, plastic closure of the large wound was accomplished by transplantation of the sub scapularis and latissimus dorsi muscles, and excision of the lower third of the scapula On a combination of viomycin and cyanacetic acid hydrazide (since he had long since become clinically resistant to the usual antimicrobials, we felt), the wound, which had been drained for about a week, healed up completely, and the bronchopleural fistulas were closed Some re expansion of the remainder of the right lower lobe became apparent on x rays of the chest The wound has remained healed for the nine months that he has been followed since the last operation The degree of medial displacement of the right shoulder girdle following partial claviculectomy, is illustrated by Figs 4 and 5

DISCUSSION

Claviculectomy, either partial or complete, is usually reserved for tumors, in fections, or trauma to this bone, or for gaining access either into the neck or the mediastinum for neurologic, vascular, or thoracic surgical procedures We found only one report of claviculectomy as an adjunct to surgical collapse of the chest wall in the literature of the past ten years, and no reference to it in any of the standard thoracic surgical textbooks Lambert,[1] in this Journal, in 1946, reported three cases of tuberculous empyema following pneumothorax, which had persisted in spite of open drainage, Schede thoracoplasty, pack, partial scapulectomy, and phrenic crush Following partial claviculectomy, two of his patients were discharged from the hospital with small draining sinuses, and one patient died of generalized disseminated tuberculosis two months after the operation These cases were treated before the appearance of streptomycin, para aminosalicylic acid, and isonicotinic acid hydrazide Since these potent antimicrobial agents have become available, the number of times that problems arise in which claviculectomy might be indicated must surely be much less than formerly That other surgeons are occasionally re-sorting to this procedure still, however, is evidenced by a personal communica-tion from Dr J D Murphy,[*] who reports two partial claviculectomies done at Oteen, North Carolina, for similar problems Both patients were reported to have done "exceedingly well, with practically no impairment of shoulder girdle function "

The technique we followed was much like Lambert's We have, of course, used separate drapes, gowns, gloves, and instruments for resecting the clavicle when a combined procedure was planned, in order to prevent the clean clavicular wound from becoming contaminated The skin incision, made over the middle half of the clavicle, is deepened through platysma muscle and periosteum down to bone The middle 6 or 7 cm of bone is stripped of periosteum and resected, the Gigli saw being a useful tool for this purpose. We have not found it necessary to resect as much of the clavicle as Lambert re ported doing After reapproximating the periosteum, and the platysma, the skin is closed without drainage Local anesthesia was used in both instances

Considering the extensive chest wall surgery carried out on both of these patients, dysfunction of the involved shoulder girdle has not been marked, in either case.

SUMMARY

Two cases of pulmonary tuberculosis are reported, in whom long-standing infected subscapular or empyema spaces, complicated in one by the presence of multiple bronchopleural fistulas, were successfully obliterated, with complete healing in both, following partial resection of the clavicle, in combination with sinus excision in one, and Schede thoracoplasty, muscle transplantation, and partial scapulectomy in the other. It is believed that the rigid clavicle occasionally prevents the shoulder girdle muscles from falling in against the chest wall or visceral pleura sufficiently to eradicate the infected space. The procedure is simple, safe, apparently effective, and associated with very little additional shoulder girdle dysfunction.

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SAMUEL J CROWE	EDWIN A. LAWRENCE
LOUIS W FRANK	EDWARD J. MCGRATH
HOWARD GRAY	ARTHUR SHIPLEY

ANNUAL MEETING NOTES

The Thirty-sixth Annual Meeting of The American Association for Thoracic Surgery was held in Miami Beach, Florida, on May 7, 8 and 9, 1956. There were 788 physicians who registered for the scientific sessions. In addition there were 125 wives present.

Following discussion over a period of two years, the Association has now employed Miss Ada Hanvey to be the Administrative Assistant. Her duties include the establishment of a permanent address of the Association at 600 South Kingshighway, St. Louis 10, Missouri. Such matters as address changes and other pertinent information should be sent directly to this address.

The following changes in the By-Laws were voted at the Executive Session:

Article II, Section 5, delete last sentence commencing "Such registered guests shall have the privilege of the floor in discussing papers if time permits."

Article VI, Add Section 5, to read as follows: "The Necrology Committee shall consist of one or more Active or Senior Members, and shall be appointed in accordance with the provisions of Article VI, Section 2 of the Constitution. Appointments to this Committee shall be for a one-year term of office. Any or all members of this Committee may be reappointed to succeed themselves. The Council may, if it so desires, appoint one of its own members to serve as Chairman of this Committee. The duties of the Necrology Committee shall be to prepare suitable resolutions and memorials upon the deaths of all members of the Association and to report such deaths at every annual meeting."

Article VII, Section 2, changed to read—"Annual dues for Active Members shall be $30.00."

Article VII, Section 3, changed to read—"Annual dues for Associate Members shall be $25.00."

A resolution was adopted requesting the American College of Surgeons to make places for three representatives of the American Association for Thoracic Surgery on the Board of Governors.

MEMBERSHIP NOTES

The Council voted to publish again the standards for election to Associate Membership as recommended by the Membership Committee in 1955.

A. Training:

1. Applicants in the United States must have a sound surgical training as evidenced by completion of the American Board of Surgery.
2. Applicants in the United States must further have met the requirements of the Board of Thoracic Surgery. Canadian applicants must have Fellowship in the Royal College of Surgeons of Canada; be qualified in thoracic surgery or have passed the Royal College examinations for certification in thoracic surgery.

B. Professional Work:

1. High moral and ethical standards are required.
2. Applicant should be established in practice in his community or in a teaching position for *at least three years following training.* His professional work must be of the highest quality.
3. The applicant should have a major interest in thoracic surgery.

4. His professional conduct should be such that he has the support of the members of the Association living in the community.
5. The applicant should show evidence of future participation in meetings by making contributions to the medical literature, advancing the knowledge of thoracic disease, and by leadership in furthering thoracic surgery in his community
6. Certification in thoracic surgery by the Board of Thoracic Surgery or by the Canadian Royal College of Surgeons *in itself does not mean fulfillment of the requirements* for election to Associate Membership. The other qualifications must be met.

C. Allied Specialists:

1. Members of the profession in allied specialties who are outstanding in their respective fields may be elected to membership in the Association according to the desirability for such specialists as determined by the Council

D. Foreign Applicants:

1. Thoracic surgeons residing outside the confines of Canada and the United States and its territories are not eligible for election to the Association except as Honorary Members

E. Exception:

1. Notwithstanding the above qualifications, in exceptional instances the Council may recommend *for election a candidate who does not meet all of the above requirements*

Honorary Membership was conferred upon Mr Philip Allison, Ch M, F R C S, Nuffield Professor of Surgery, Oxford, England

The following changes in membership status were voted by the Association during its annual Executive Session

The following Active members were elevated to Senior Status:

Clarence Bird
Emile Holman
William A Hudson
Robert M Janes
Richard H Meade
Herman Moersch
James D Murphy
James W Nixon
Dudley E Ross
Edward S. Welles

The following Associate members were elected to Active membership.

Oscar Auerbach
Theodore Badger
Henry T. Bahnson
Ivan D. Baronofsky
Edward B Benedict
Harrison Black
Louis H. Bosher
Robert I Carlson
Max G. Carter
Sheldon E Domm
William H Falor
Egbert H. Fell
Edward A Gaensler
Paul Geary
Orville F. Grimes
Lew A Hochberg
William A Hopkins
Elgie K. Johnson
Hollis E Johnson
Richard King
Harold A. Kipp
Robert Klopstock
Floyd J. Lewis
Clarence W. Lillehei
Saul A. Mackler
Edgar P Mannix
Gabriel Seley
Arthur J Vorwald
Paul F. Ware

The following candidates were elected to Associate membership:

Richard H. Adler, Buffalo, N. Y.
John W. Bell, Sunmount, N. Y.
Francis N. Cooke, Miami, Fla.
Philip Crastnopol, Kew Gardens, N. Y.
Frederick S. Cross, Cleveland, Ohio
Alfred M. Decker, Jr., Sunmount, N. Y.
Sanford W. French, III, San Francisco, Calif.
Francis S. Gerbasi, Long Beach, Calif.
Frank Glenn, New York, N. Y.
Allen E. Greer, Oklahoma City, Okla.
Ormand C. Julian, Chicago, Ill.
Frederick C. Kittle, Kansas City, Kan.
Irving M. Madoff, Brookline, Mass.
Robert W. Newman, Knoxville, Tenn.
Andrew G. Morrow, Rockville, Md.
S. Gwin Robbins, Memphis, Tenn.
Raleigh R. Ross, Austin, Tex.
John M. Salyer, Denver, Colo.
Hawley H. Seiler, Tampa, Fla.
George W. B. Starkey, Brookline, Mass.
Frederick H. Taylor, Charlotte, N. C.
William E. Van Fleit, Emory University, Ga.
George R. Walker, Sudbury, Ont., Can.
Harry E. Walkup, Oteen, N. C.
John L. Wilson, Beirut, Lebanon

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